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A CRITICAL ANALYSIS OF STANDARD METHODS OF ESTIMATING HEART SIZE FROM ROENTGEN MEASUREMENTS*

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THE problem of devising a reliable method of estimating heart size by roentgen-ray mensuration in order to determine the presence or absence of cardiac enlargement has interested cardiologists and roentgenologists since the introduction of clinical roentgenology four decades ago. Various roentgen-ray measurements have been advocated and standards of normal established in each instance. In addition, prediction tables for normality based on body measurements have been elaborated, particularly by Hodges and Eyster[5,6] and more recently by Ungerleider and Clark.[8] In spite of a considerable literature on this subject and some general agreement as to the relative worth of these various methods, the situation remains in general unclarified and unstandardized as a routine clinical procedure. It is our experience that many roentgenologists in their routine diagnostic work are giving too much weight to certain classical but, nevertheless unreliable, standards of normality and thereby, in some instances, misinforming the clinician as to the presence of cardiac enlargement.

The great difficulty in arriving at satisfactory standards for distinguishing between normal and abnormal heart size by roentgen-ray measurements lies in the wide range of normal values and the overlapping of measurements for normal and abnormal hearts. This is clearly illustrated in Figure 1. The very wide range in the size and shape of the normal heart is evident and it is obvious that a small heart could enlarge considerably before it would exceed the size of the larger normal hearts. Actually the principal difficulties in establishing a reliable and accurate borderline between normality and cardiac enlargement are caused by enlargement occurring in small hearts and the fact that some normal hearts, particularly those transversely placed in the chest, have exceedingly large measurements. In view of the confusion which exists in regard to cardiac mensuration it seemed most desirable

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to have a comparative analysis of these various standard methods of estimating normal heart size and to attempt to evaluate their relative worth and reliability from a clinical standpoint.

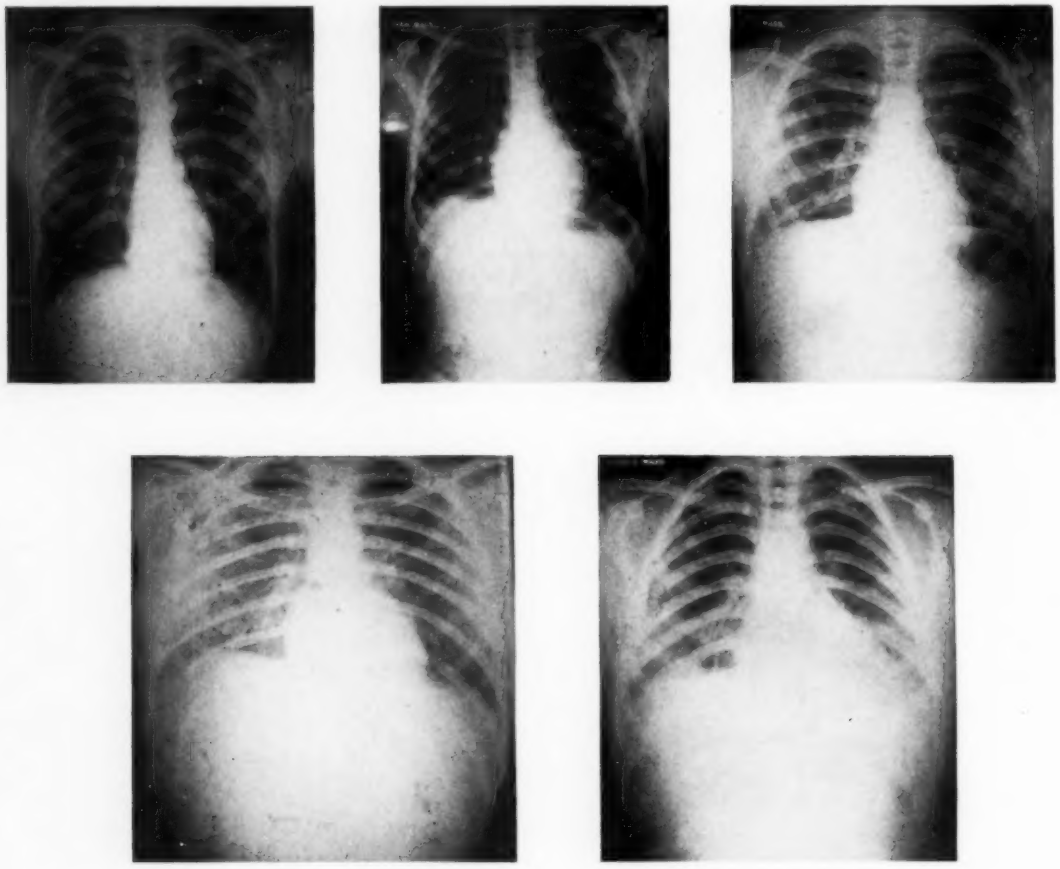

FIG. 1. Variation of the size and shape of the normal heart.

MATERIAL

Our material consists of 150 males and 50 females with normal hearts. Their ages ranged from fifteen to seventy years with the majority in the latter half of the second, and in the third, fourth and fifth decades of life (Table 1). This age range is important for it seems to us unreliable to derive standards of normality based entirely on young students. Throughout this study orthodiascopy was the roentgenographic method employed. In each instance the following measurements were determined on each individual: (1) the transverse diameter of the heart; (2) the area of the frontal cardiac silhouette by a planimeter; (3) the heart volume by the Rohrer-Kahlstorf formula;* (4) the cardiothoracic ratio; (5) and (6) the deviation from the predicted normal of the transverse heart diameter and the frontal cardiac area as determined by the formulae of Hodges and Eyster in nomograms published by Kurtz.[7]

ANALYSIS OF MATERIAL

The basic data are summarized in Tables II, III, VII, and VIII, and show the spread and distribution of the various actual measure-

* In 50 of the younger males this measurement was omitted.

ments and the deviations from the predicted normal in the 200 cases. From these it would appear desirable that standards of heart size based on actual roentgen-ray measurements should distinguish between males and females since, in the latter, the heart is consistently smaller than in males. It will be noted, however, that this is largely compensated for in estimates based upon body size, as indicated by the results from prediction tables.

ORTHODIAGRAPHIC HEART MEASUREMENTS

Transverse Diameter of the Heart. From our results the transverse diameter of the heart lies between 9.5 and 13.9 cm. in 93 per cent of normal individuals. In only 4 per cent does the transverse diameter of the heart exceed the latter figure and in only 8 per cent is this measurement greater than 13.4 cm. In 90 per cent of males the transverse diameter is normally less than 13.4 cm. In females the limits are less, with 94 per cent of women having a transverse diameter between 9.0 and 12.4 cm. Since the point which demarcates normal heart size from cardiac enlargement is of primary interest, the important upper limits for transverse heart diameters are 13.4 cm. for males and 12.4 cm. for females. It seems safe to conclude, therefore, that the very great majority of individuals having transverse diameters greater than these upper limits will have cardiac enlargement. We are planning a further study of this group to determine the approximate percentage of normal and abnormal cases having measurements of transverse diameter greater than these upper limits.

TABLE I

Age Distribution

Age	15–19	20–29	30–39	40–49	50–59	60–70
Males	45	35	24	30	12	4
Females	8	19	7	11	5	0
Total	53	54	31	41	17	4

Frontal Cardiac Area. Examination of our data on the frontal cardiac area shows that in 90 per cent of normal individuals this measurement will fall between 80 and 119 sq. cm. Further, in 95 per cent of normal males this measurement will not exceed 119 sq. cm. In females, again, the normal heart is smaller, with no cardiac area greater than 109 sq. cm. and 92 per cent of the women having areas less than 104 sq. cm. Thus with a frontal cardiac area greater than 119 sq. cm. in males and 104 sq. cm. in females there is a 10 per cent chance or less of such a heart being of normal size.

TABLE II

Normal Distribution of Actual Heart Measurements

Transverse Heart Diameter															
Centimeters	8.5 8.9	9.0 9.4	9.5 9.9	10.0 10.4	10.5 10.9	11.0 11.4	11.5 11.9	12.0 12.4	12.5 12.9	13.0 13.4	13.5 13.9	14.0 14.4	14.5 14.9	15.0 15.4	
College Males	0	1	2	3	8	3	11	9	4	7	1	1	0	0	
Miscellaneous Males	0	0	2	2	10	14	20	13	11	15	7	2	4	0	
Females	0	5	4	9	7	8	10	4	2	1	0	0	0	0	
Total	0	6	8	14	25	25	41	26	17	23	8	3	4	0	
Frontal Cardiac Area															
Square Centimeters	60 64	65 69	70 74	75 79	80 84	85 89	90 94	95 99	100 104	105 109	110 114	115 119	120 124	125 129	130 134
College Males	0	0	1	2	6	8	7	12	3	5	3	3	0	0	0
Miscellaneous Males	0	0	1	0	8	2	8	19	13	14	15	12	4	2	2
Females	1	1	1	5	12	10	5	7	4	4	0	0	0	0	0
Total	1	1	3	7	26	20	20	38	20	23	18	15	4	2	2
Heart Volume															
Cubic Centimeters	250 299	300 349	350 399	400 449	450 499	500 549	550 599	600 649	650 699	700 749	750 799	800 849	850 899	900 949	950 999
Miscellaneous Males	0	0	1	5	10	11	25	19	17	3	4	3	0	2	0
Females	1	2	10	6	20	7	3	0	1	0	0	0	0	0	0
Total	1	2	11	11	30	18	28	19	18	3	4	3	0	2	0

Heart Volume. In approximately 90 per cent of normal individuals the heart volume lies between 350 and 700 cc. with 8 per cent exceeding this upper limit. Except for 9 per cent all normal males have heart volumes of less than 750 cc. while again in 92 per cent of the females the upper limit is considerably less, 550 cc. Thus in the majority of individuals a heart volume of more than 750 cc. in males and more than 550 cc. in females would indicate cardiac enlargement.

Comparison of the Transverse Diameter, the Frontal Cardiac Area and the Heart Volume. Since the measurements discussed above are actual roentgen-ray heart measurements in contrast to heart size determinations based on correlative methods, it is advisable to compare these at this point. To us there seem to be three criteria of importance in evaluating the relative clinical worth of any method of estimating heart size, i.e. (1) the narrower the range (the less spread) of normal values, the more desirable the method, (2) the smaller the technical errors in the method, the more reliable the measurement, and (3) the fewer the technical difficulties and the less time consuming the method, the more practical it becomes clinically.

In Table III are tabulated the upper normal limits for each measurement and the percentage of normal hearts which can be expected to exceed these limits. In addition, this table shows for each measurement the total spread of the normal values and the narrowest possible spread to include approximately 90 per cent of the cases. When differences in the units of measurement are taken into consideration there is still a significant difference in total spread which favors the transverse heart diameter when both sexes are considered together. However, there is relatively little difference in total spread when these measurements are analyzed according to sex, which emphasizes again the importance of differentiating the sexes in actual mensuration. When 90 per cent of the cases are examined there is a surprising consistency throughout except for the heart volume in females which appears to have a slightly narrower concentrated range of values.

TABLE III

Upper Limits and Spread of Actual Normal Measurements

		Upper Limits of Normal	Percent of Cases Greater Than Upper Limits	Total Spread	Spread of 90% of Cases
Tranverse Heart Diameter	Males	13.4 cm.	10%	5.9 cm.	3..9 cm.
	Females	12.4 cm.	6%	4.4 cm.	3.4 cm.
	All Cases	13.4 cm.	8%	5.9 cm.	3.9 cm.
Frontal Cardiac Area	Males	119 sq. cm.	5%	64 sq.cm.	39 sq.cm.
	Females	104 sq. cm.	8%	49 sq.cm.	34 sq.cm.
	All Cases	119 sq. cm.	4%	74 sq.cm.	39 sq.cm.
Heart Volume	Males	749 cc.	9%	599 cc.	349 cc.
	Females	549 cc.	8%	449 cc.	249 cc.
	All Cases	699 cc.	8%	699 cc.	349 cc.

This advantage, however, becomes less significant when the other two criteria are considered, for there is a considerable error associated with volumetric determinations and technically the procedure is somewhat complicated and time consuming. Consequently, from the standpoint of narrowest spread there can be no question that transverse heart diameters yield equally if not more favorable results than either the cardiac area or the heart volume.

As to the degree of technical error associated with these measurements the transverse diameter would appear to be the least subject to error. Table IV in which orthodiagrams were done on the same individual on three different days was an attempt to determine the consistency of results which might be expected from these three measurements and thereby to estimate the degree of error in their clinical application. It

is evident here and it has been shown elsewhere[1] that the maximum error in determining the heart volume is considerable (±10 per cent for normal sized hearts and ±15 per cent for enlarged hearts).

In respect to the transverse diameter of the heart and the cardiac area our findings indicate that when hearts of normal size are examined in this manner there is a slight, although not significant, difference in the average error which favors the former. When large hearts are measured on successive days we find no increase in error for determinations of the transverse heart diameter whereas in cardiac areas the error increases, significantly at times. The increased error in estimating cardiac areas and the fact that occasionally an isolated high percentage of error is encountered can be explained, we believe, by differences in completing the inferior and superior portions of the cardiac silhouette which are fused with the diaphragm and great vessels respectively. In a previous report[1] we showed that the error in this respect when the same individual completes the silhouette may range from ±1 to ±5 per cent. Further when different individuals complete the same silhouette the percentage error may range as high as ±12. There can be no doubt that since actual mensuration of transverse heart diameters contains no variables more consistent results can be expected.

It is true theoretically that the transverse diameter will fail to detect cardiac enlargement of the left upper border, common in mitral stenosis, which will be included in the determination of the frontal cardiac area. However, even in pure mitral stenosis it is only in advanced disease when enlargement is obvious that there is sufficient

TABLE IV

Three Successive Measurements of Same Individual

	Tranverse Heart Diameter				Frontal Cardiac Area				Heart Volume			
Determination	I	II	III	%	I	II	III	%	I	II	III	%
Normal Heart	9.1	9.5	9.5	4%	73	73	78	7%	411	390	449	15%
"	9.5	9.5	9.4	1%	79	79	78	1%	388	375	345	12%
"	10.0	10.0	9.7	3%	89	95	91	7%	456	491	465	8%
"	10.8	10.9	11.2	4%	93	98	98	5%	491	507	514	5%
"	11.0	11.0	11.1	1%	98	97	96	2%	642	589	577	11%
"	11.3	11.3	11.6	3%	107	106	108	2%	585	625	594	7%
"	11.3	12.1	11.8	7%	100	105	114	11%	636	610	702	15%
"	11.5	11.4	11.4	1%	109	107	113	6%	568	581	595	5%
"	12.1	11.6	11.6	4%	116	114	115	2%	598	601	577	4%
"	12.5	12.9	12.5	3%	111	112	101	11%	620	642	589	9%
"	13.6	13.4	13.5	1%	122	120	120	2%	807	742	749	9%
"	13.8	14.5	14.0	5%	120	126	123	5%	829	883	884	7%
Average				3%				5%				9%
Heart Disease	12.0	11.9	11.7	3%	110	110	107	3%	633	631	628	1%
"	13.8	13.6	13.7	1%	130	122	114	14%	759	817	698	17%
"	15.8	15.4	15.2	4%	158	155	147	7%	1195	1139	1039	15%
"	20.0	19.7	20.6	5%	204	208	233	14%	1609	1584	1905	20%
Average				3%				9%				13%
Combined Average				3%				7%				11%

dilatation of the left auricle or pulmonary conus in the frontal silhouette to make a significant difference in the frontal area. In 4 cases of mitral stenosis we found that deviations from the predicted normal for both cardiac area and transverse diameter indicated cardiac enlargement in 3 cases, and in one both indicated normal size. Consequently we doubt if this advantage of the frontal area is as great in practice as it would appear to be theoretically.

over long periods of time. Actually it is only in an exceptional instance that in any given studied case a change in the level of the diaphragm distorts the transverse diameter significantly and in these instances this artifact is usually obvious clinically since obesity or abdominal distention from some cause or other is invariably the responsible factor.

There can be little argument that the transverse diameter is simpler and more ap-

TABLE V

Maximum Change With Respiration

Measurement	Transverse Heart Diameter				Frontal Cardiac Area			
Phase of Respiration	Quiet	Deep Insp.	Deep Exp.	% Change	Quiet	Deep Insp.	Deep Exp.	% Change
Vertical Heart	9.1	10.2	11.2	23%	73	81	74	11%
"	10.0	10.7	12.4	24%	98	111	110	13%
Mid Position	10.7	9.2	10.8	17%	81	89	80	11%
"	11.2	11.5	12.3	10%	101	117	102	16%
Transverse Heart	13.1	12.9	14.7	14%	105	118	111	12%
"	13.0	11.4	13.1	15%	94	92	104	13%
Average				17%				13%

A frequent criticism of the transverse heart diameter is that changes in the position of the diaphragm cause considerable distortion of this measurement whereas the cardiac area is little affected. This objection, however, is not valid practically. Table v shows that extreme positions of the diaphragm can distort both these measurements significantly, with the cardiac area altered, on the whole, somewhat less than the heart diameter. Further, it is evident from Table iv that from day to day there can be little distortion of the heart from this source in the same individual. We have repeatedly noted the consistency of transverse diameters in following private cases with normal or fully compensated hearts

plicable routinely than the determination of the cardiac area or the heart volume. From the literature we were led to believe, and we expected to find here that the cardiac area was definitely superior to determinations of transverse heart diameters. However, after an unbiased examination of our data we must conclude that the transverse heart diameter, although it, too, is open to criticism and error, is equally, if not more reliable than the frontal cardiac area in determining heart size, and that it has certain advantages in respect to inherent error and clinical applicability.

CORRELATIVE METHODS

Of the somatic measurements which have

been studied, heart size correlates best with either body weight or body surface area. The correlation with body height is less close, and age in adults has little value as a correlative factor. It is now generally agreed that the best approach to the problem of measuring heart size in order to determine the presence or absence of cardiac enlargement in an individual case is to have standards of normal which are based on one or more body measurements proved to have a correlation with heart size. The existence or absence of cardiac enlargement in any single instance is then judged on the basis of predicted normal heart size (average normal heart size) for individuals with similar body measurements. A combination of body weight and body height has so far proved to be the most satisfactory correlative factor.* Standards of normal heart size by prediction tables have been established by Hodges and Eyster for orthodiagraphic measurements, and these tables are the basis of most studies of this nature.

Cardiothoracic Ratio. This method of estimating the presence or absence of cardiac enlargement was the first of the correlative methods and is still widely employed by both clinicians and roentgenologists despite the fact that its reliability has been justifiably questioned. Beside the fact that internal chest diameters correlate poorly with heart size, it has been recently shown by Ungerleider and Clark[8] that there exists a definite mathematical fallacy in attempting correlations between transverse heart and chest diameters.

A popular misconception in regard to the cardiothoracic ratio is illustrated in a recent text on heart measurements which states rather unequivocally that there is little doubt of cardiac enlargement if this ratio is over 50 per cent. The questionable accuracy of such a statement is manifest when of our normal material (Table VI), 15 per cent of males and 26 per cent of females had cardiothoracic ratios of over 50 per cent. This method is particularly fallible by indicating cardiac enlargement in individuals with normal but transversely placed hearts and, vice versa, by indicating normal heart size in individuals with hearts vertically placed in the thorax when cardiac enlargement exists. To obviate the former inaccuracy, if this method is to be used, 55 per cent would appear to be a more reliable upper limit of normality. The fallacy of this method in failing to detect cardiac enlargement in small hearts is less easily remedied. Further, we cannot see that this method has any advantage over merely determining the actual transverse diameter of the heart provided the latter is correlated either objectively or subjectively with body size. Besides these theoretical and real objections there exist, and will be

TABLE VI

Normal Distribution of Cardiothoracic Ratio

Per cent	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58
College Males	0	1	7	3	4	5	2	3	5	2	5	3	6	4	0	0	0	0	0	0	0
Miscellaneous Males	1	0	1	4	1	10	10	11	6	9	8	10	10	4	5	4	3	0	1	1	1
Females	0	0	0	1	1	2	3	6	3	5	8	3	5	4	0	3	2	2	1	0	1
Total	1	1	8	8	6	17	15	20	14	16	21	16	21	12	5	7	5	2	2	1	2

* At present a combination of height and weight has proved to be the most satisfactory correlative index of body size although from the preliminary studies[2] the correlation of heart size and body build appears even more promising. The establishment of a combination of somatic measurements by which general types of body build can be expressed is essential for the practical application of such a correlation. No simple index for expressing body build exists and we are at present making anthropometric studies in an attempt to establish a practical index for body build and thereby to give us an opportunity to test its correlation with heart size.

TABLE VII

Normal Distribution Based On Prediction Tables

Per cent Deviation From Predicted Normal		-31 -35	-26 -30	-21 -25	-16 -20	-11 -15	-6 -10	-1 -5	0 0	+1 +5	+6 +10	+11 +15	+16 +20	+21 +25	+26 +30
Frontal Cardiac Area	College Males	3	8	8	11	10	5	5	0	0	0	0	0	0	0
	Miscellaneous Males	1	0	3	7	9	24	18	4	13	13	5	2	0	1
	Females	0	1	4	7	9	7	9	1	7	3	0	1	0	1
	Total	4	9	15	25	28	36	32	5	20	16	5	3	0	2
Transverse Heart Diameter	College Males				1	8	6	14	3	10	6	2	0		
	Miscellaneous Males				2	7	9	31	0	22	22	6	1		
	Females				4	7	9	15	2	8	4	1	0		
	Total				7	22	24	60	5	40	32	9	1		

discussed presently, correlative methods of estimating cardiac enlargement which are far more reliable and no more difficult technically than the cardiothoracic ratio. On the whole, we believe that the present general reliance upon the cardiothoracic ratio for detecting cardiac enlargement leads to avoidable errors and that it would be best were it discarded as a routine procedure, particularly since an equally simple and much more reliable method is available.

Predicted Frontal Cardiac Area. In Eyster's[3] series of 100 individuals with normal hearts, 25 per cent of the cardiac areas exceeded ±10 per cent deviation from the predicted normal and of these 9 per cent had a greater deviation than +10 per cent. Kurtz[7] in another series of 500 normal cases found that 88 per cent of his normal cases had a frontal cardiac area which fell within the limits of ±10 per cent deviation from the predicted normal and only 3 per

TABLE VIII

Spread of Normal Predicted Measurements

Deviation from Predicted Normal		+10% to −10%		Less Than −10%		More Than +10%		Total Spread
		Number of Cases	Percent of Total	Number of Cases	Percent of Total	Number of Cases	Percent of Total	
Frontal Cardiac Area	Males	82	55%	60	40%	8	5%	−35% to+30%
	Females	27	54%	21	42%	2	4%	−30% to+30%
	Total	109	54.5%	81	40.5%	10	5%	−35% to+30%
Transverse Heart Diameter	Males	123	82%	18	12%	9	6%	−20% to+20%
	Females	38	76%	11	22%	1	2%	−20% to+15%
	Total	161	80.5%	29	14.5%	10	5%	−20% to+20%

cent of the remaining 12 per cent exceeded a +10 per cent deviation. Our results (Tables VII and VIII) using this method showed that 45 per cent of the normal cardiac areas lay beyond a ±10 per cent deviation with no significant difference between the sexes (45 per cent males, 46 per cent females). The wide range by this method is evidenced by the fact that values were scattered between +30 per cent and −35 per cent deviation from the predicted normal. However, only 5 per cent had a deviation which was greater than +10 per cent, again with little difference between males and females (males 5 per cent, females 4 per cent). It is evident that there is a considerable discrepancy in the degree of concentration of normal cardiac areas between the limits of a ±10 per cent deviation in these three series of cases. We are unable with any degree of certainty to account for this discrepancy between the series of Eyster and Kurtz and of our own. It is possible that in part this discrepancy may be explained by the fact that we have completed the upper border of the cardiac outline less generously than was illustrated by Kurtz. The completion of the cardiac outline in our material was based on a study of heart models made in this laboratory from actual clinical and pathological material.

In our own group we found that the cardiac areas in all but 10 of 50 college students (a rather tall group on the whole) ranged between a −10 per cent and −35 per cent deviation from the predicted normal and of the remaining 10, not one had a plus deviation. Although measurements by all the methods were on the whole slightly smaller in the college group and the extreme high normal values were fewer, there was no such marked discrepancy as was manifested by the method under discussion. Unless the heart size lags behind body growth as evidenced by height and weight, one must conclude that the prediction tables are responsible.* The inclusion of this group, however, does not distort the results as much as might at first be expected. Excluding this college group, 34 per cent of cases still exceed a ±10 per cent deviation and 7 per cent instead of 5 per cent exceed the predicted normal by more than + 10 per cent. Although both the series of Eyster and Kurtz show approximately a one to one distribution of the plus and minus deviations, in our results the latter is weighted by a two to one ratio without the college group, and by a three to one ratio if it is included. From our results we must conclude that the prediction table for frontal cardiac area tends to overestimate the size of the normal heart and consequently that it may fail in many instances to detect cardiac enlargement when it exists.

Predicted Transverse Heart Diameter. In regard to the predicted transverse heart diameter (Tables VII and VIII), we found that 80 per cent of normals were included between a ±10 per cent deviation with only 6 per cent exceeding a + 10 per cent deviation. Estimating from the charts of Eyster and Kurtz† we find that in the former 84 per cent and in the latter 91 per cent of normal cases fell within a ±10 per cent deviation with 8 per cent and 3 per cent respectively of cases exceeding a +10 per cent deviation. Our findings, then, by this method correspond satisfactorily with Eyster's series. In constructing prediction tables based upon teleroentgenographic measurements, Ungerleider and Clark[8] found that 80 per cent of normals fell within a ±10 per cent deviation with 12 per cent exceeding a +10 per cent deviation. It is obvious that by this method there is general agreement between the results of Eyster, Ungerleider and Clark, and ourselves. As a generalization it can be said that 80 per cent of normal hearts will fall between a ±10 per cent deviation from the predicted normal transverse diameter and that not more than 10 per cent will exceed a +10 per cent deviation. The distribution in our series between the plus and minus de-

* Our planimeter has been checked and found accurate and since the silhouettes were completed by the same person, all technical errors seem to be eliminated.

† Actual figures not given.[7]

viations for transverse diameters is fairly well balanced with a three to two ratio, the same as in the group studied by Eyster. In Kurtz's series the plus and minus deviations were almost balanced equally, with a five to four ratio.

Comparison of the Two Prediction Methods. It may be noted that we did not test these methods by using them as an index for distinguishing between individuals with and without heart disease as was done by both Eyster and Kurtz. We question the validity of such a test for judging the reliability of any method of estimating heart size, since heart disease and cardiac enlargement are not necessarily synonymous. In uncomplicated coronary disease, for example, there is often no cardiac enlargement, and even in mitral stenosis there may be sufficient dilatation of the posteriorly placed left auricle and of the right ventricle to be discerned in any measurement made in an anterior projection of the heart shadow only in advanced cases. Furthermore, a normally small heart may enlarge considerably and still be within established normal limits.

Cardiac enlargement is always most easily demonstrated in measurements derived from an anterior silhouette in those individuals whose heart disease affects primarily the left ventricle. In a previous study we hoped to remedy this obvious difficulty with methods using only one plane by investigating heart volume determinations. Our study in this regard proved disappointing, due largely to the high percentage of the technical error of the method when applied practically and we were forced to conclude that until perfected, volumetric determinations have no advantages over simpler methods of determining heart size.

Table VIII shows clearly that there is a high percentage of concentration of values (81 per cent) between a ± 10 per cent deviation from the predicted normal in the case of the transverse heart diameters which is not evident when the frontal cardiac area is employed (55 per cent). Since there is no difference between the two so far as values exceeding a $+10$ per cent deviation (5 per cent) are concerned, the gain in concentration made by employing transverse heart diameters is made in the values having less than a -10 per cent deviation. This, plus the fact that deviations from predicted normal transverse heart diameters have a much less total spread than the cardiac areas, indicates that there is more likelihood of detecting cardiac enlargement in the normally small hearts by employing prediction tables based on transverse heart diameters. The consistency of our results with those of others in regard to predicted heart diameters in contrast to the discrepancies associated with predicted cardiac areas further favors the reliability of the former. To these factors favoring the use of transverse heart diameters over frontal cardiac areas in prediction tables must be added, since an actual heart measurement is applied in this method, the conclusion which we made above, that of the actual measurements the transverse diameter is also favored. From our results one must conclude, therefore, that deviations from predicted normal transverse diameters fulfill most satisfactorily both the theoretical and practical aspects of the problem of cardiac mensuration.

GENERAL DISCUSSION

It is evident that the problem of establishing a clinical method of measuring heart size so that the presence or absence of cardiac enlargement in every instance can be established unequivocally has yet to be solved, and, due to the biological nature of the problem with the wide variations of normality, it is very unlikely that such an ideal will ever be attained. We believe that actual heart measurements alone have a limited value and that they are most satisfactory in observing the changes in heart size which occur as an individual case is followed periodically. In this regard our findings indicate that of the actual measurements studied, the transverse diameter

of the heart is in general as reliable as any direct measurement and much more suitable from the viewpoint of general clinical applicability. We feel certain, however, that actual mensuration alone, except in obviously normal or enlarged hearts, is not the most reliable method for determining the presence or absence of cardiac enlargement in an individual case.

Of the available methods for determining cardiac enlargement the most sound are those based upon correlations between body size and heart size. It has been shown that the size of the normal heart depends in a large measure upon the body build and the size of the individual and only by correlative methods of determining heart size can this very important fact be utilized. We consider the use of prediction tables as the most reliable available method of establishing the existence of cardiac enlargement. Although Eyster, Kurtz and others feel that the most reliable results are obtained with the frontal cardiac area, our results lead us to conclude that by employing the transverse diameter of the heart, and a deviation from the predicted normal of greater than +10 per cent used as an index for the existence of cardiac enlargement, more reliable results are obtained. This conclusion permits the general adoption of this method since the transverse diameter of the heart is a simple measurement and is at present widely employed in some capacity or other for estimating heart size. We wish to emphasize that the use of prediction tables is a no more complicated procedure technically than the routine methods now in general usage since, other than the height and weight of the individual and the transverse heart diameter, no other data are necessary. Furthermore, from the viewpoint of general clinical applicability prediction tables now exist for measurements made from standard 6 foot teleroentgenograms as well as from orthodiagrams.

It should be pointed out that although a majority of individuals with significant cardiac enlargement can be defined by cardiac mensuration, there is no reliable method of determining how many individuals with slight or moderate cardiac enlargement are included within the established normal limits.

In borderline cases the transverse heart of a heavy individual often presents great difficulties since such hearts, normally, have usually large measurements and the presence of cardiac enlargement is thus difficult to establish. There is need for a better correlation standard to express body build than body weight and height, and if the present somatic standards can be improved more reliable results may be expected.

Consequently, it should be more widely recognized that although heart measurements are helpful as an aid to diagnosis, they are not infallible and considerable judgment must be employed in their interpretation. The opinion of an experienced observer, particularly one familiar with the clinical aspects of the case, as to the existence of cardiac enlargement after roentgen examination of the heart and lungs, is more valuable by itself than a heart measurement, although the latter may be of considerable importance in forming a final opinion.

CONCLUSIONS

1. A comparison of transverse heart diameters, frontal cardiac areas, and heart volumes in 200 normal hearts (150 males and 50 females) leads us to conclude that the transverse diameter of the heart compares favorably with the other actual heart measurements and is the most satisfactory from the clinical standpoint.

2. We believe that the cardiothoracic ratio is not sufficiently reliable to warrant the wide usage which it now enjoys and that it should be discarded in view of the fact that it has been superseded by a more accurate and an equally simple correlative method.

3. We have found that prediction tables offer the best approach to the problem of

determining whether cardiac enlargement exists in an individual case. Our results indicate that the use of the transverse heart diameter and its deviation from the predicted normal is as yet the most reliable and the most applicable clinically of the existent methods. The use of prediction tables is as simple a method of estimating heart size as the determination of the cardiothoracic ratio and can be routinely applied as easily. Since prediction tables for the transverse diameter based either on orthodiagraphic or teleroentgenographic measurements are now available and since the transverse diameter of the heart is now widely employed routinely in some capacity or other in determining heart size, prediction tables based on transverse heart diameters may well be adopted generally and accepted as the standard clinical procedure for cardiac mensuration, until a better correlation is introduced.

4. Cardiac mensuration is a valuable but not infallible aid in determining the presence or absence of cardiac enlargement. It should not be accepted unreservedly, however; one must combat such a tendency as in any other laboratory procedure in which mathematical limits are established. Heart measurements must be interpreted with judgment and with the realization that exceptions to the established limits exist. Of particular importance in evaluating these measurements is the appreciation of the importance of body build and the position of the heart in the thorax in determining the size and shape of the normal heart. Consequently, rather large measurements may indicate a relatively small heart for individuals of a certain habitus whereas similar or even smaller measurements may signify considerable cardiac enlargement in individuals of a different body build. This factor is too often overlooked in the routine roentgenological examination of the heart.

Prediction tables based upon teleroentgenograms may be obtained from Dr. Harry Ungerleider of the Equitable Life Assurance Society of New York. If orthodiagraphic measurements are used, tables or nomograms for prediction purposes may be found in Kurtz's book or a cardiac slide rule developed in the diagnostic laboratories of the Equitable Life Assurance Society and manufactured by the Picker X-ray Corporation of New York City may be used.

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ADDENDUM

Further discussion by Dr. Comeau in answer to questions on this subject at the annual meeting of the American Heart Association in Cleveland, Ohio, on May 30, 1941:

I thoroughly agree with Dr. Kurtz (Chester M. Kurtz, Madison, Wisconsin) that the cardiothoracic ratio should be given up, and that a prediction method should be utilized as a routine procedure for cardiac mensuration.

From the practical aspect, I think we all agree that the cardiac area is a more complicated procedure than the transverse diameter, and it is true that the more complicated the procedure, the less likely it is that it will be used widely.

There are several important sources of error in estimating cardiac areas. The main one is the completion of the superior and inferior borders of the cardiac shadow, which are hidden by the great vessels above and the diaphragm below. I know that my results on the cardiac area dif-

fer from those found by Dr. Kurtz, and I am quite convinced that the reason they do differ is because I have been less generous in completing the hidden borders of the cardiac silhouette.

It is also true that there is an individual error that I have proved on myself by measuring the cardiac area from tracings of an original orthodiagram on different dates. I, too, like Dr. Kilgore (Eugene S. Kilgore, San Francisco, Calif.) have found occasional differences of as high as 10 per cent, and I have also checked against other individuals completing the same orthodiagram and found errors as high as 15 per cent. It may be that an occasional check will show an error, as was commented upon, as high as 25 per cent.

In regard to the question of Dr. Master (Arthur M. Master, New York City) concerning measurements of left ventricular hypertrophy, I know of no measurements other than by Dr. Robb's (George P. Robb, New York City) roentgen method of outlining the left ventricular cavity with opaque material thereby enabling one to measure the thickness of the left ventricular wall. This, of course, is not a very practical method for routine use.

In conclusion, I would like to leave this impression: when a cardiac measurement is employed, one should appreciate that it is merely an aid in diagnosis and that it should not be accepted too literally. The acceptance, for example, of a cardiothoracic ratio of over 50 per cent as indicating cardiac enlargement is actually not uncommonly encountered and unquestionably has led to many erroneous diagnoses of heart disease. Actually, the most important factor in estimating heart size is the impression obtained by a reliable and experienced roentgenologist. Cardiac measurement should be utilized as an aid in diagnosis after a thorough roentgen examination but should not be accepted as the final authority in determining whether a heart is enlarged or not.

(I agree fully with the remarks presented by Dr. Comeau in the discussion above. Paul D. White.)

A RADIOGRAPHIC DEMONSTRATION OF THE CIRCULATION THROUGH THE HEART IN THE ADULT AND IN THE FOETUS, AND THE IDENTIFICATION OF THE DUCTUS ARTERIOSUS*

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From the Nuffield Institute for Medical Research
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THE application of radiology to the study of the circulation has recently been made possible by the advance of X-ray cinematography combined with intravascular injection of radio-opaque substances. In this paper an account is given of the results of some of the radiological experiments we have undertaken, which demonstrate the circulation through the heart and great vessels adjoining it, both in the adult‖ and in the fœtus. The transitional stage between the fœtal and adult types of circulation is also demonstrated, and the identification of the ductus arteriosus, as it appears in the films, is discussed in considerable detail. The main object of our experiments was a study of the ductus arteriosus, but in the course of our research we also obtained a demonstration of the circulation, and it is with this aspect of our work that we are more specially concerned in this paper. Films showing the adult circulation have already been made by others (*e.g.*, Janker, Böhme), but so far as we know this is the first radiological demonstration of the fœtal circulation.

For the study of the adult circulation, sheep, cats, and rabbits have been used, and for that of the fœtal circulation and the transitional stage the subjects have been sheep fœtuses, varying from 100 to 145 days in age (full term = 147 days), delivered by Cæsarean section. In the study of the fœtal circulation, although for technical reasons the fœtus could not be actually *in utero* during the period of recording, we have tried to preserve intra-uterine conditions so far as possible, leaving the umbilical cord attached to the placenta and covering the snout of the fœtus with a rubber bag containing amniotic fluid, in order to prevent respiration.

The operations involved in the experiments were conducted under various forms of anæsthesia. The minor procedure of opening the vein to be injected was carried out either under nembutal, in the case of cats, or under urethane, in the case of rabbits and adult sheep, while for the sheep fœtuses a local anaesthetic (novocaine or borocaine) was used, supplementing in many instances the effect of urethane injected into the mother prior to Cæsarean section. The Cæsarean sections were for the most part performed under a general anæsthetic in the form of urethane, but latterly we have used both spinal and local anæsthetics (duracaine and borocaine) with the object of obtaining more normal conditions in the fœtus than may be the case in a urethanised animal.

The opaque media used have been thorotrast, uroselectan B, and perabrodil, sub-

‖ As this paper is concerned with the circulatory apparatus alone, we have extended the meaning of the term "adult" to connote any animal in which both the ductus arteriosus and the foramen ovale have ceased to function as blood channels. The term has thus been applied to both new-born lambs and also, in its ordinary sense, to mature animals of various species.

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stances which mix freely with the blood stream, and lipiodol, an iodised oil, which is carried along in the blood as individual drops. We will refer later to the results obtained with this last substance. All the media were injected at approximately body temperature. Thorotrast, even in large quantities, did not appear to affect the animals, but its complete elimination from the blood by the reticulo-endothelium of the liver and spleen is relatively slow. Hence, for the repeated injections employed in some of our experiments, this substance was not suitable owing to the persistence of the shadow in the blood stream. Uroselectan B is rapidly eliminated by the urinary tract, but did not seem to be well tolerated by the fœtus in the doses that we had to use. Perabrodil in a 50 per cent solution was also followed by symptoms, but the same medium in 35 per cent strength appeared to be well tolerated.

The sites of injection have been the external jugular and femoral veins of the adult, and the external jugular and umbilical veins—and in two cases the femoral artery—of the fœtus.

Two methods of recording have been used: (1) direct X-ray cinematography, *i.e.*, taking ordinary radiographs at a speed of 3–4 per second on long bands of X-ray film, 5 inches wide, each picture being 5 inches square;* (2) indirect X-ray cinematography, *i.e.*, photographing the image on the fluorescent screen with a 16 mm. cine camera at a speed of 25 pictures per second.† The latter method is specially useful in illustrating the passage of individual drops of lipiodol within the blood stream, but the direct method gives far more detail and, hence, greater value from the point of view of analysis. The illustrations in this paper are all taken from films produced by the direct method; the diagrams, on the other hand, were constructed from tracings of films produced by both methods.

* The apparatus for this technique was built by the Cambridge Instrument Company for one of us (A.E.B.) with the assistance of a grant from the Medical Research Council, and was briefly described in *Brit. J. Radiol.*, N.S. viii, 682, 1935.

† Two sets of apparatus were used for this method, one designed by Dr. Russell Reynolds and the other of home construction, which was more convenient for the fœtal work.

ADULT CIRCULATION

In the adult, all venous blood entering the heart, whether from the superior or from the inferior vena cava, passes through the right auricle and the right ventricle, leaves the heart by the pulmonary valve and flows along the pulmonary arteries to the lungs (Figs. 1A and 1C; Diagrams A and B, p. 687). The return flow of oxygenated blood passes from the lungs along the pulmonary veins to the left auricle, through the left ventricle to the aortic valve, and so out from the heart into the aorta (Figs. 1B and 1D; Diagram C, p. 687). (It will be noted in the diagram that only one vessel, the brachio-cephalic artery, is shown branching from the aorta towards the upper segment. In the cat there are generally two vessels, the innominate and left subclavian arteries, but in the sheep one appears to be the rule.)

FŒTAL CIRCULATION

Bearing in mind the course of the circulation through the heart of the adult, we turn now to the circulation in the fœtus. In order to meet the very different requirements of the fœtal circulation, two bypasses are provided for shortcircuiting the pulmonary system: (1) the ductus arteriosus, and (2) the foramen ovale. As will be shown later, the purpose of the ductus arteriosus is to enable venous blood entering the heart from the superior vena cava to pass directly to the descending aorta (and thence to the placenta) without traversing the lungs, while the object of the foramen ovale is to allow re-oxygenated blood from the placenta to pass from the right auricle to the left side of the heart and thus directly to the aorta and the branches which spring from the arch.

Flow from Superior Vena Cava. Venous blood entering the heart from the superior vena cava passes through the right auricle and right ventricle to the pulmonary valve and trunk, where the stream divides, one

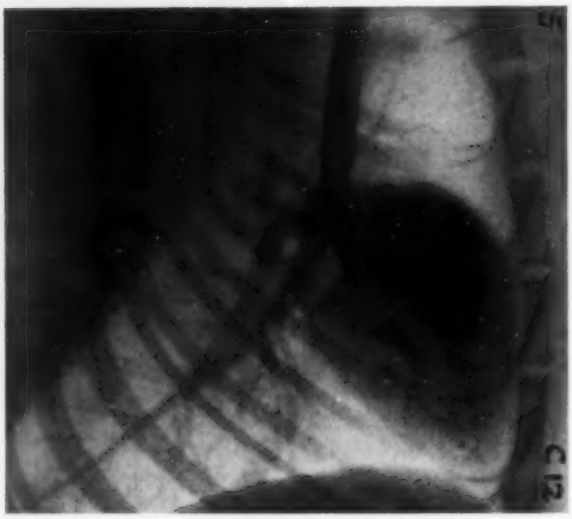


FIG. 1A. (Adult cat.) Injection passing down the superior vena cava, through the right side of the heart, to the pulmonary arteries.

portion passing through the ductus arteriosus (the identification of which is discussed in some detail below) to the descending aorta and the remainder passing through the pulmonary arteries to the lungs (Fig. 2A; Diagram D, p. 687). In due course, the return flow from the lungs is

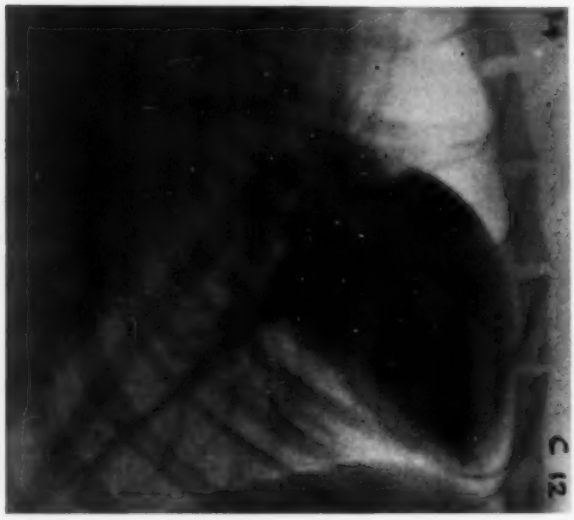


FIG. 1B. 3 seconds after A, the contrast medium returns from the lungs, by the pulmonary veins, to the left side of the heart, and passes into the aorta and its branches.

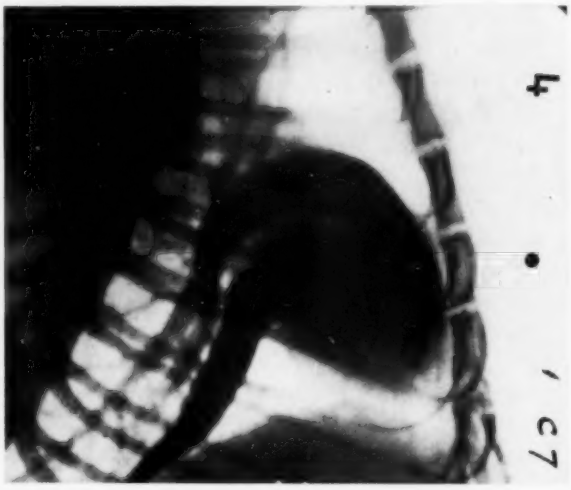


FIG. 1C. (Adult cat.) Injection passing up the inferior vena cava, through the right side of the heart, to the pulmonary arteries.

seen passing along the pulmonary veins to the left auricle and left ventricle, and thence through the aortic valve to the aorta and brachio-cephalic artery (Fig. 2B; Diagram E, p. 687), taking the same course as the return flow from the lungs in the adult (Diagram C). Diagram F, p. 687, (constructed by superimposition of Diagrams D and E) shows the relation of the ductus arteriosus to the other vessels.

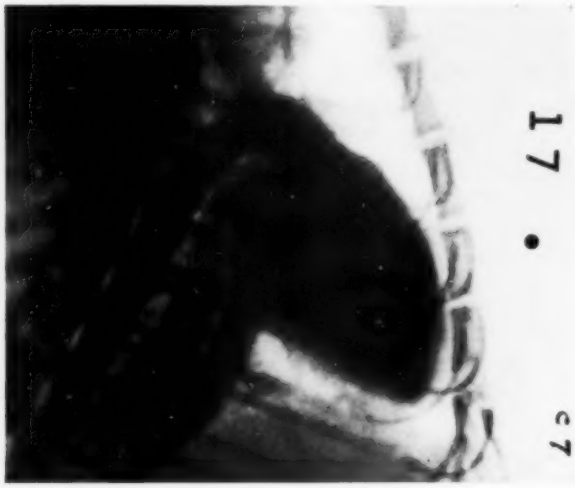


FIG. 1D. $4\frac{1}{3}$ seconds after C, the injection has returned from the lungs and is passing through the left side of the heart to the aorta and its branches.

The Ductus Arteriosus. In a paper in this Journal (*Brit. J. Radiol.*, N.S. xi, 570, 1938) we identified the ductus arteriosus in the new-born lamb as a vessel of fine calibre, the shadow of which apparently formed a junction between that of the pulmonary arterial system and that of the aorta. Since that time, and owing to considerable improvements in technique, we have found that we were wrong. The shadow which we then described as that of the ductus is really that of a branch of one of the pulmonary arteries. The actual shadow of the ductus is quite other. In order to rectify this mistake we must discuss in some detail the identification of the ductus, which, as will be shown, is a vessel of considerable size, approximating to that of the aorta itself.

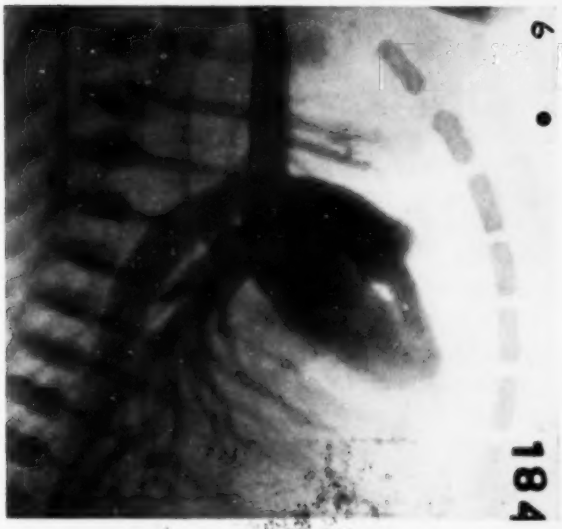


Fig. 2A. (Sheep fœtus.) Injection passing down the superior vena cava, through the right side of the heart to the pulmonary trunk, and thence to the pulmonary arteries and (*via* the ductus arteriosus) to the descending aorta. Note the conus arteriosus and pulmonary valve.

In any radiograph in which the vessels in the vicinity of the heart are all filled with an opaque material, such as thorotrast, the ductus is almost entirely merged in a complex shadow derived from the following structures: the superior vena cava, the right auricle, the pulmonary trunk, the ductus arteriosus, the aortic arch, the root of the brachio-cephalic artery, and possibly that of the pulmonary arteries. This complex shadow is seen framed in a rectangle in Fig. 3, and it will be realised that it is

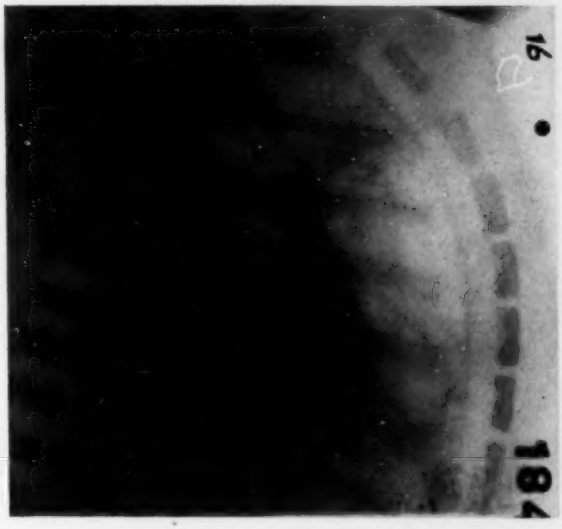


Fig. 2B. 3½ seconds later, the contrast medium is seen returning from the lungs, by the pulmonary veins, to the left side of the heart (the ventricle is incompletely filled) and passing to the aorta and brachio-cephalic artery.

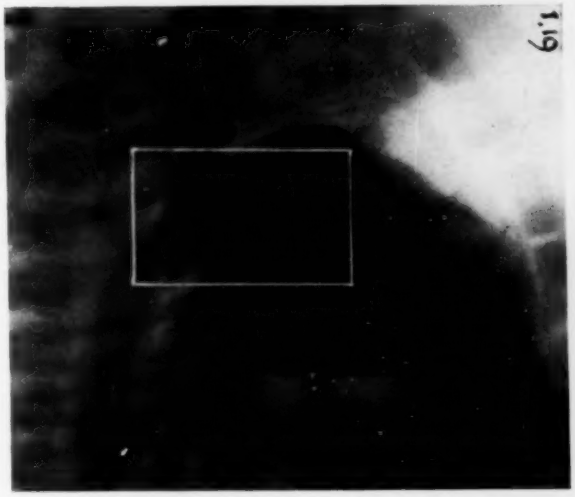


Fig. 3. (Sheep fœtus.) In this radiograph the vessels in the vicinity of the heart are all filled with contrast medium. The area framed in a rectangle shows the complex shadow in which the ductus arteriosus is concealed.

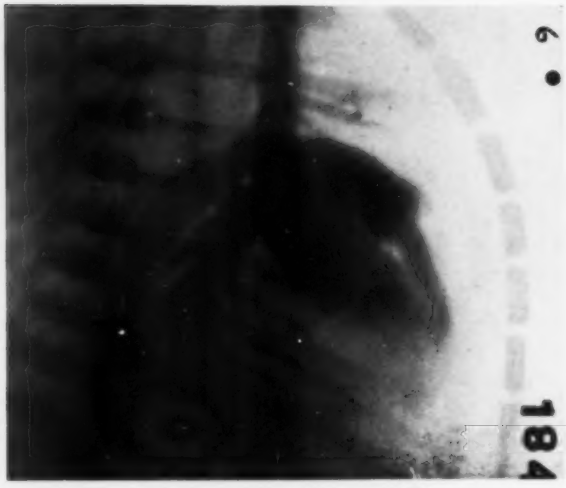


FIG. 4A. (Sheep fœtus.) Injection passing down the superior vena cava, through the right side of the heart to the pulmonary trunk, and thence to the pulmonary arteries and (*via* the ductus arteriosus) to the descending aorta. The contrast medium has not yet reached the left side of the heart.

impossible to single out the ductus in this picture. Some special technique is required for locating the exact site of the vessel and for visualising it without the complication of superimposed shadows. For this purpose we have used three types of experiment:—

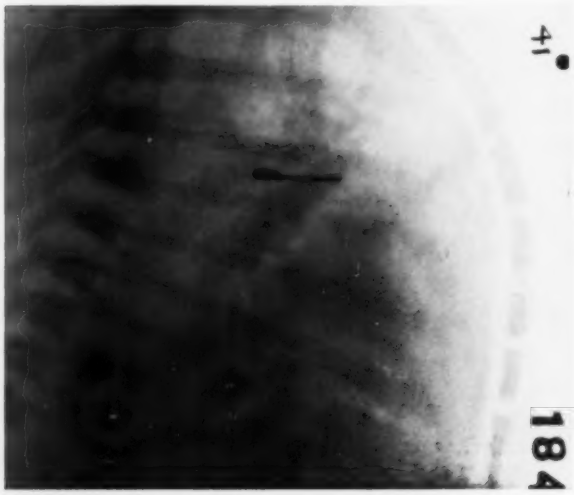


FIG. 4B. Radiograph of the same animal as that shown in 4A, taken after the thorax had been opened and a piece of wire sewn on to the ductus arteriosus. The contrast medium injected previously has not yet been entirely eliminated from the blood, and still faintly outlines the vessels.

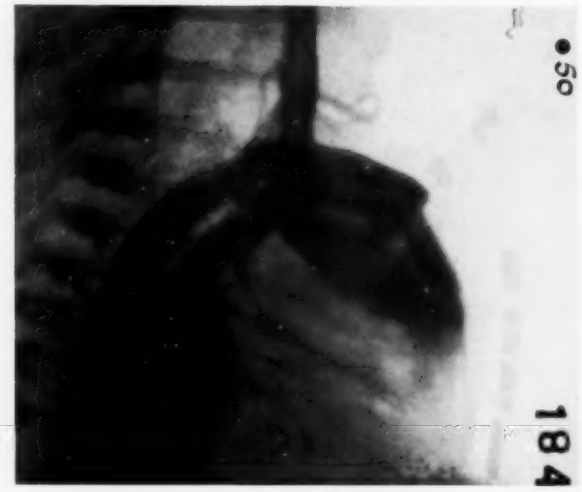


FIG. 4C. Radiograph taken 3 seconds after 4B, showing an injection similar to that shown in 4A, with the piece of wire in position on the ductus.

(1) Making an injection after having labelled the ductus by sewing on to it a piece of wire.

(2) Injecting a small quantity of contrast medium fairly rapidly, so that this passes through the cardio-vascular system

FIG. 4D. 4 seconds later, the injection shown in 4C is seen to have passed on from the right heart and to be returning from the lungs, along the pulmonary veins, to the left side of the heart, passing thence through the aortic valve to the aorta and brachio-cephalic artery. (*Cf.* the curve of the aortic arch with that composed of the pulmonary trunk, ductus, and descending aorta, shown in 4C.)

en masse, showing the various vessels and chambers of the heart in succession.

(3) Making a retrograde injection, which passes from the femoral artery up the aorta.

We will discuss the results of these experiments in order.

(1) After a preliminary injection of thorotrast had been recorded in its passage through the heart from the superior vena cava (Fig. 4A), the thorax was quickly opened on the left side and a piece of wire of the same length as the ductus was sewn on to this vessel along the side facing the aperture in the thorax. The thorax was then closed and Fig. 4B shows the wire in position. It will be seen that the thorotrast previously injected has not yet been entirely eliminated and still faintly outlines the vessels. A second injection was then made (Fig. 4C). It will be seen that the wire is almost lost in the general shadow and that it lies at right angles to the superior vena cava. (It is of interest to note that the opening of the thorax has not seriously disturbed the anatomical picture (*cf.* Fig. 4A). Throughout the experiment the umbilical cord was intact and a nose bag in position, so that the lungs were not expanded.) In Fig. 4D, four seconds later, the thorotrast has been completely cleared from the right heart and the ductus, and is seen returning from the lungs by the pulmonary veins to the left heart and passing to the aorta and brachio-cephalic artery. The relation of the wire on the ductus to the aortic arch is well seen.

(2) What has already been said indicates the possibility of seeing the ductus uncomplicated by the shadow of other vessels if, after an injection *via* the superior vena cava, a radiograph is taken at the moment when the contrast medium has just passed on from the superior vena cava, leaving this vessel empty, and is filling the right heart and vessels leading from it, but has not yet reached any of the vessels connected with the left side of the heart. Such a moment is illustrated in Fig. 5. The course of the ductus, as it branches from the pulmonary trunk, is well seen, but its exact point of origin and its anastomosis with the aorta cannot be accurately determined. The slight break in the natural curve of the aorta shows the approximate site of the anastomosis. In many of our records, however, the site of origin of the ductus and its entry into the aorta can be determined with considerable accuracy by superimposition of tracings of the different vessels taken from radiographs in which the right and left sides of the heart are shown in succession. Diagram F, p. 687, has been constructed in this way. There is a slight variation in the length of the ductus in different animals, and also in its point of entry into the aorta—hence the little differences that may be noted in the diagrams and in the radiographs in which the ductus is shown.

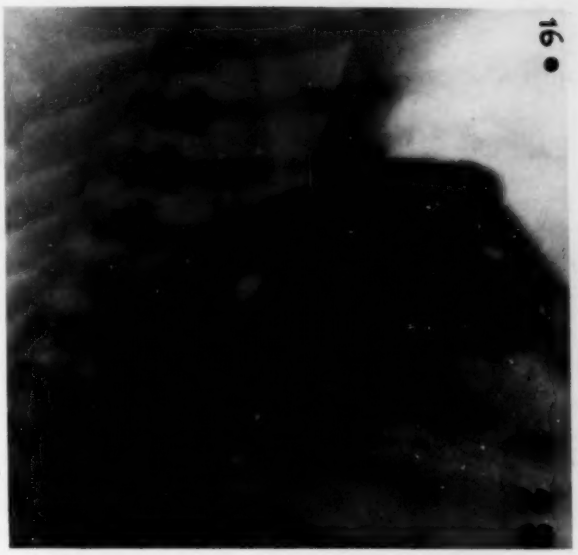


Fig. 5. (Sheep fœtus.) Radiograph showing the relation of the ductus arteriosus to the pulmonary trunk, the pulmonary arteries and the descending aorta. The contrast medium, which has entered the heart from the superior vena cava, has just been cleared from this vessel, and it has not yet reached any of those directly connected with the left side of the heart, with the result that much of the shadow which generally confuses the picture of the ductus is absent. The site of origin of the ductus in the pulmonary trunk cannot be accurately determined here, but the approximate position of its anastomosis with the aorta is indicated by the slight break in the natural curve of that vessel.

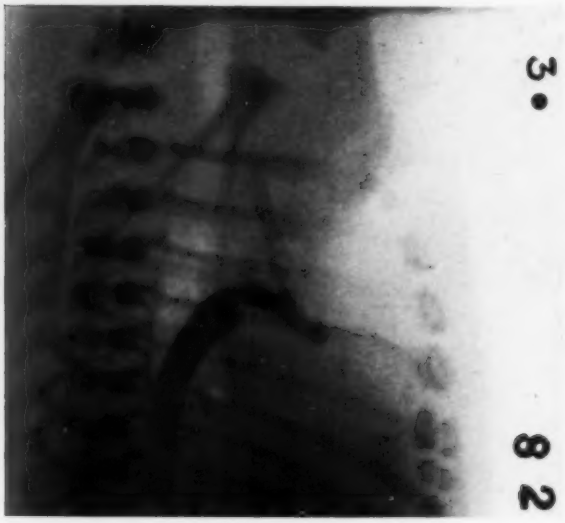


FIG. 6A. (Sheep fœtus.) Retrograde injection passing up the aorta, round the arch to the aortic valve and up the brachio-cephalic artery. Note (just below the aortic arch) the contrast medium trying to make its way through the ductus

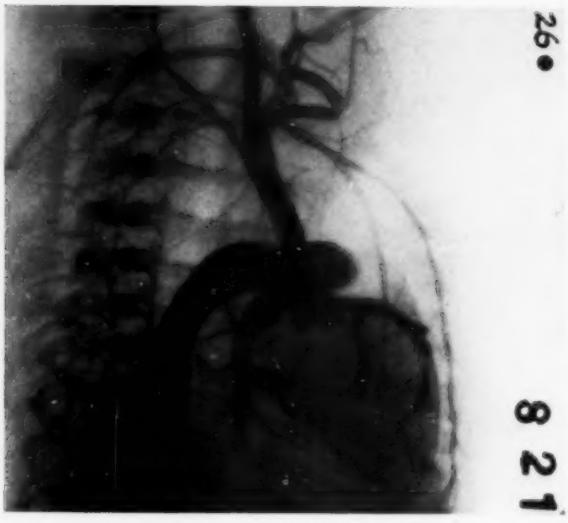


FIG. 6C. 3¼ seconds after 6B, the injection has now passed right through the ductus, almost to the pulmonary valve, and has been turned back to pass down the pulmonary artery. Note the aortic valve and the coronary artery.

(3) Any ambiguity as to the exact site of the ductus and the relation of its shadow to those of the aorta and pulmonary trunk has, however, been removed by an experiment in which an injection of thorotrast was made into the femoral artery in the direction of the heart. The fœtus, a young animal of approximately 100 days, was for

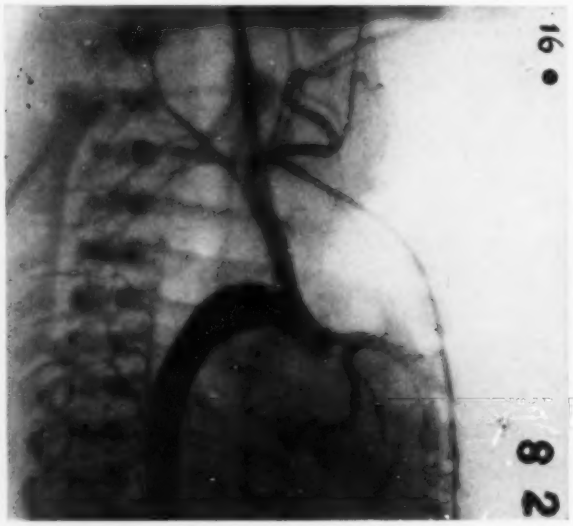


FIG. 6B. 4¼ seconds later, the injection has passed considerably farther up the brachio-cephalic artery and the arteries branching from it, while one of the coronary arteries is also filled. The contrast medium has passed rather farther along the ductus than in 6A.

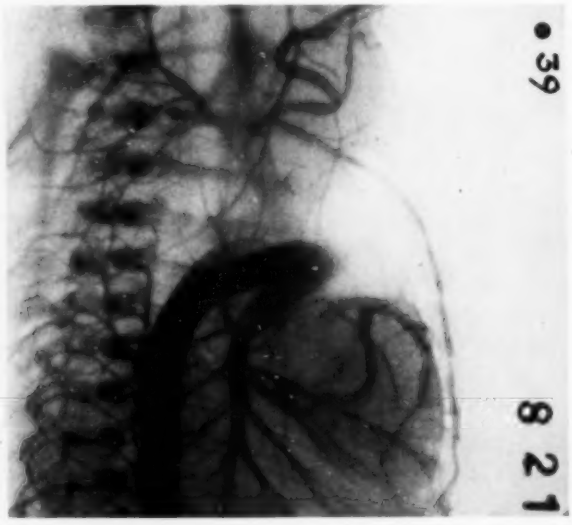


FIG. 6D. 4¾ seconds after 6C, a feeble pulsation of the left heart has momentarily cleared the ascending aorta and partially cleared the arch and brachio-cephalic artery, thus leaving a well-defined picture of the ductus uniting the pulmonary trunk and descending aorta.

various reasons in a moribund condition, its heart beating only feebly and from time to time. The umbilical cord had been divided and the nose bag removed. The pressure of the injection was greater than that of the heart beat, and consequently the thorotrast ascended the aorta and was only prevented from entering the heart by the aortic valve. In Fig. 6A the thorotrast is seen to have passed up the aorta, round the arch to the aortic valve, while a small quantity is seen pushing its way up the brachio-cephalic artery. Just below the aortic arch the contrast medium is seen trying to make its way through the ductus, but passing only a very little way into this vessel. In Fig. 6B the shadow in the brachio-cephalic artery is more clearly marked, and one of the coronary arteries, springing from a sinus of Valsalva, is excellently shown. The thorotrast has pushed its way through the ductus across the arch of the aorta rather farther than in Fig. 6A, and in Fig. 6C it has made its way right through this vessel to the pulmonary trunk and has been turned back just before reaching the pulmonary valve and passed down one of the pulmonary arteries. In Fig. 6D a feeble pulsation of the left heart has momentarily cleared the thorotrast from the ascending aorta, and almost cleared it from the arch and the brachio-cephalic artery, with the result that we have a picture of the ductus, uniting the pulmonary trunk and the descending aorta, virtually uncomplicated by superimposed shadows.

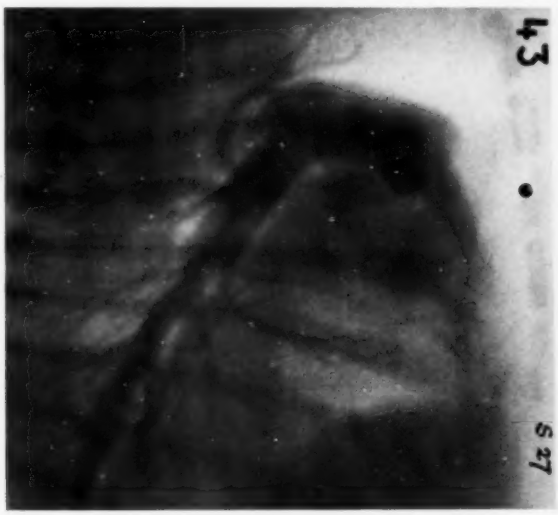


FIG. 7A. (New-born lamb.) Radiograph showing the pulmonary trunk and arteries. Note the pointed stub springing from the pulmonary trunk at the site of the ductus. The vessel is functionally closed, and allows no blood to pass to the descending aorta.

FIG. 7B. 1⅔ seconds later, the right heart is empty, and the contrast medium is seen returning from the lungs by the pulmonary veins to the left side of the heart, and passing out to the aorta and brachio-cephalic artery. There is no indication of any back-flow from the aorta through the ductus to the pulmonary trunk.

In addition to the above methods which afford an *anatomical* identification of the ductus arteriosus, the patency or closed state of the vessel can be determined by a *functional* test, by observing the course taken by the blood. Since the ductus enters the aorta at a point distal to the origin of the brachio-cephalic artery (see Diagram F, p. 687) a shadow seen in the descending aorta, without a corresponding shadow being visible in the brachio-cephalic artery, indicates the stream has passed to the aorta through the ductus from the *right* side of the heart. Had it come through the aortic valve from the left side of the heart, the brachio-cephalic artery would also be seen.

Flow from the Superior Vena Cava. In the

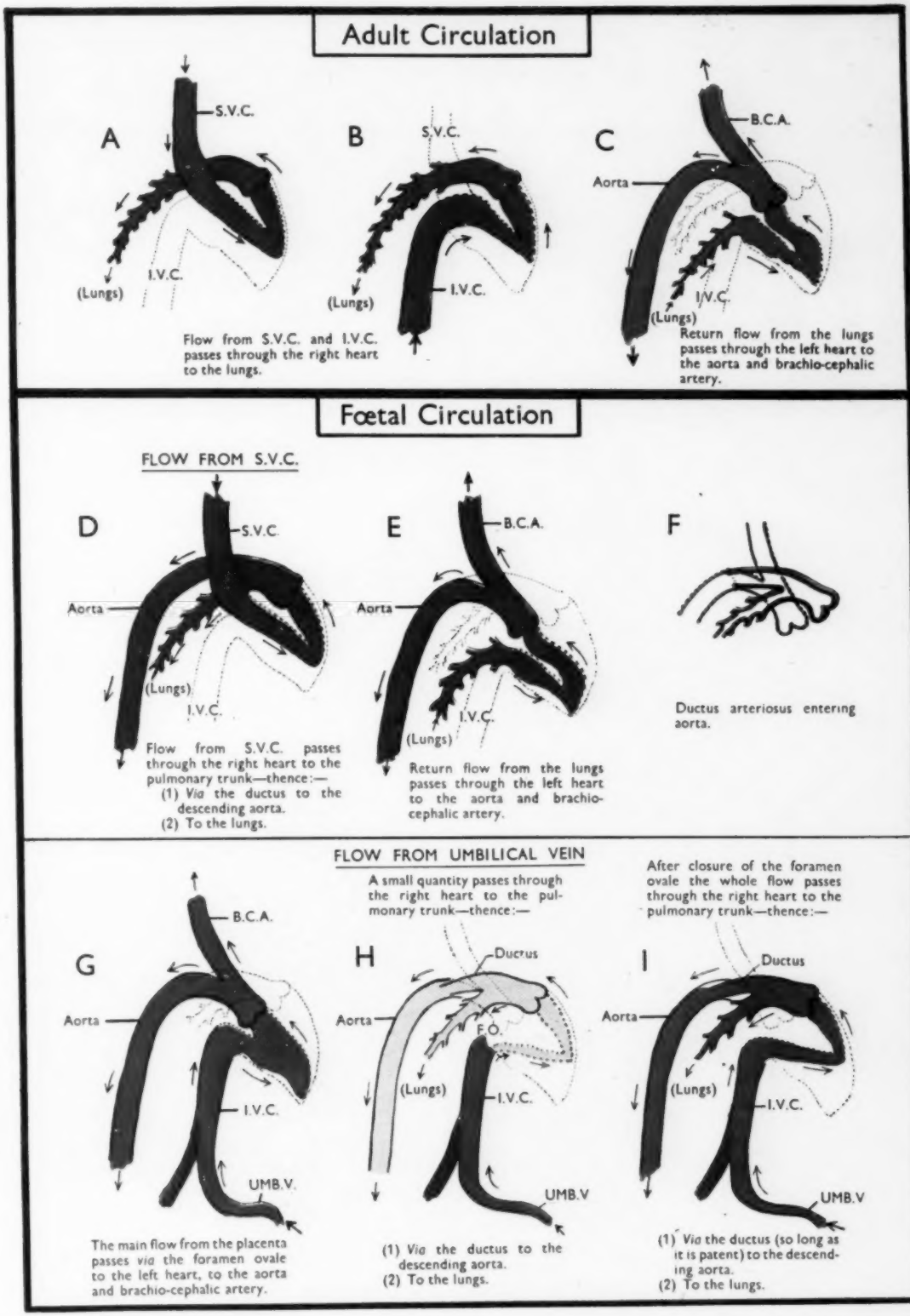
FIG. 8A. (Sheep fœtus.) Radiograph showing an injection passing up the inferior vena cava to the right auricle. The greater part of the contrast medium passes through the foramen ovale to the left side of the heart, and thence to the aorta and brachio-cephalic artery. A small quantity, however, is also seen in the right side of the heart, the pulmonary trunk, and the ductus.

records of all our injections entering the heart *via* the superior vena cava, there has been no instance of any shadow being visible in the brachio-cephalic artery until after the opaque blood had returned to the left side of the heart from the lungs or other sources. It appears that none of the blood from the superior vena cava passes through the foramen ovale, but that the whole flow from this source passes through the right side of the heart only. Hence, a shadow appearing in the descending aorta immediately after injection of opaque medium *via* the superior vena cava indicates that the ductus arteriosus is patent. When the ductus is closed, so far as our experiments show, the whole flow from the superior vena cava passes through the pulmonary arteries to the lungs, as in the case of the adult (Diagram A, p. 687).

Closure of the Ductus Arteriosus. According to our records, the ductus closes functionally shortly after delivery (within 5 minutes in one experiment), and in a number of our radiographs we find a stub projecting from the pulmonary trunk at the site of the ductus (Fig. 7A). This appears to be the blind end of the functionally closed vessel. In Fig. 7B (about two seconds later) which shows the return flow from the lungs to the left heart and the filling of the aorta and brachio-cephalic artery, it will be noted that the whole of the right side of the heart is clear and that the stub of the ductus can no longer be seen, *i.e.*, there is no suggestion that any of the aortic flow is passing back into the ductus. A more detailed account of our findings on this and other points will be published at a later date.

Flow from the Inferior Vena Cava (Umbilical Vein). In the fœtus, the inferior caval flow is composed of oxygenated blood returning to the heart from the placenta *via* the umbilical vein, and venous blood from the lower segment. We have not as yet traced the course taken by this venous blood, our experiments having been confined to the demonstration of the flow from the umbilical vein. The main portion of this stream, after entering the right auricle, passes through the foramen ovale (the exact site of which we have not yet identified on the films) to the left auricle and left ventricle, and is seen immediately leaving the

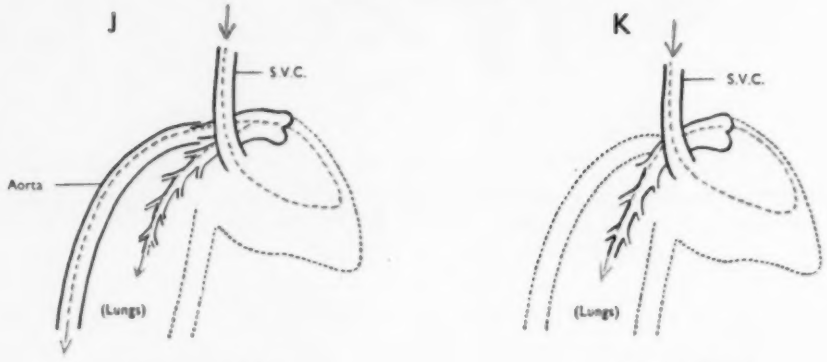
FIG. 8B. This radiograph, taken 1 second later, shows more clearly the main portion of the flow from the inferior vena cava passing through the left side of the heart to the aorta and brachio-cephalic artery, and the small quantity passing through the right side of the heart to the pulmonary arteries.


Adult Circulation
A
S.V.C.
I.V.C.
(Lungs)
Flow from S.V.C. and I.V.C. passes through the right heart to the lungs.
B
S.V.C.
I.V.C.
(Lungs)
C
B.C.A.
Aorta
I.V.C.
(Lungs)
Return flow from the lungs passes through the left heart to the aorta and brachio-cephalic artery.
Fœtal Circulation
FLOW FROM S.V.C.
D
S.V.C.
Aorta
(Lungs)
I.V.C.
Flow from S.V.C. passes through the right heart to the pulmonary trunk—thence:—
(1) Via the ductus to the descending aorta.
(2) To the lungs.
E
B.C.A.
Aorta
I.V.C.
(Lungs)
Return flow from the lungs passes through the left heart to the aorta and brachio-cephalic artery.
F
Ductus arteriosus entering aorta.
FLOW FROM UMBILICAL VEIN
G
B.C.A.
Aorta
I.V.C.
UMB.V.
The main flow from the placenta passes via the foramen ovale to the left heart, to the aorta and brachio-cephalic artery.
A small quantity passes through the right heart to the pulmonary trunk—thence:—
H
Ductus
Aorta
F.O.
(Lungs)
I.V.C.
UMB.V
(1) Via the ductus to the descending aorta.
(2) To the lungs.
After closure of the foramen ovale the whole flow passes through the right heart to the pulmonary trunk—thence:—
I
Ductus
Aorta
(Lungs)
I.V.C.
UMB.V
(1) Via the ductus (so long as it is patent) to the descending aorta.
(2) To the lungs.

Tracings from indirect X-ray cine films (16 mm.) showing the different courses taken by globules of Lipiodol, injected *via* the external jugular and umbilical or femoral veins, in the fœtus and in the adult (sheep).

Course of flow from the S.V.C.

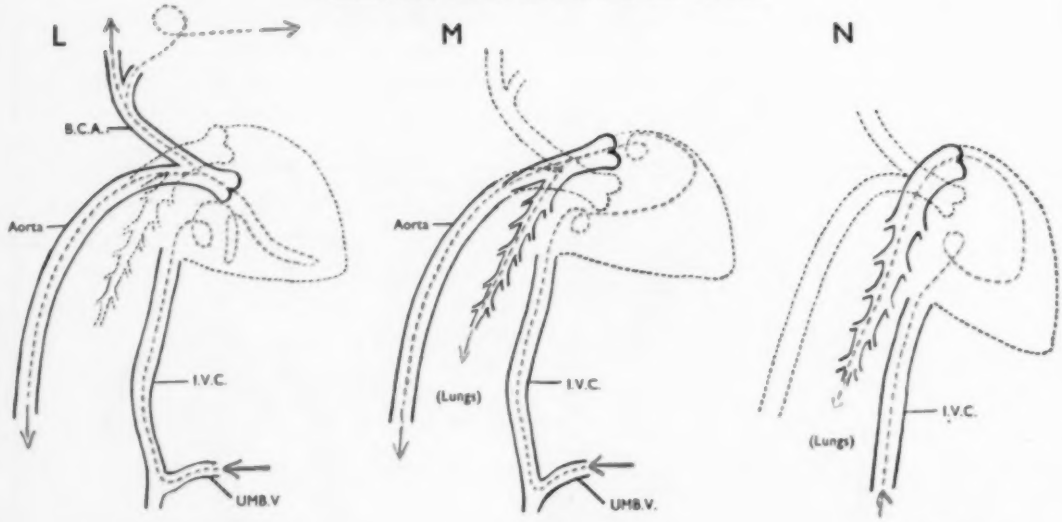
(Injection *via* external jugular vein)



Fœtus (S.14)
Ductus patent

Adult (S.3)
Ductus closed

Course of main flow from the I.V.C.



Fœtus (S.18)
Foramen ovale patent
(Injection *via* umbilical vein)

Fœtus (S.18)
Foramen ovale closed
Ductus patent
(Injection *via* umbilical vein)

Adult (S.2)
Foramen ovale and
ductus closed
(Injection *via* femoral vein)

heart by the aortic valve and flowing along the aorta and brachio-cephalic artery (Fig. 8A; Diagram G, p. 687). In addition, however, a certain quantity is detached from the main stream in the right auricle and is carried through the right atrio-ventricular (tricuspid) valve to the right ventricle and pulmonary trunk (Figs. 8A and 8B; Diagram H, p. 687). Hence, we see a relatively faint shadow in the right side of the heart and pulmonary arteries, *i.e.*, the flow is comparatively small in relation to the main stream through the left side of the heart and aortic valve. When the foramen ovale closes, the whole of the flow that enters the heart from the inferior vena cava is taken through the right side of the heart to the pulmonary trunk, passing thence through the pulmonary arteries to the lungs, and—so long as it is patent—through the ductus arteriosus to the descending aorta (Diagram I), giving the same picture as that of the flow from the superior vena cava seen in Diagram D. When the ductus also closes, the whole flow passes to the pulmonary arteries and adult circulation is established (Diagram B). See p. 687.

It will be seen, therefore, that just as the presence of a shadow in the descending aorta, immediately after an opaque medium has entered the heart from the superior vena cava, indicates the patency of the ductus arteriosus (as explained above), so also the presence of a shadow in the brachio-cephalic artery, immediately after contrast material has entered the heart from the inferior vena cava, indicates the patency of the foramen ovale.

Injections of Lipiodol. The use of lipiodol as a contrast medium gives rise to possible fallacies in the interpretation of the normal blood flow. The individual drops of this substance tend to adhere to the walls of the vessels. Moreover, they cannot pass the capillaries, except in extremely fine division (as in Böhme's films), and consequently the arterial vessels—particularly the pulmonary arteries—become obstructed, so that it is only in the initial period that these drops are likely to give a relatively true representation of the flow.

Injections of lipiodol (emulsified in a mortar with about one quarter of its volume of normal saline) have been recorded chiefly by the indirect method of X-ray cinematography at a speed of 25 pictures per second. The course of the globules through the heart and vessels can be watched on the screen, but their passage is too rapid for accurate observation even at the slowest rate of projection. For our analysis, therefore, we have projected the individual frames of these films and traced the progress of the globules frame by frame. It is in this way that Diagrams J—N, p. 688, have been constructed. As already indicated, a true representation of the natural flow can be relied upon only before the peripheral circulation has been obstructed. In order to avoid possible fallacies, therefore, the tracings from which the diagrams were made illustrate the course taken by the earlier drops of the injections.

It will be seen from the diagrams that the course of the flow from both the superior and the inferior vena cava demonstrated by the use of this substance entirely confirms the results of the analysis of films of injections of thorotrast and similar media recorded by the direct method.

One small point that is not indicated in the Diagram should be mentioned, *i.e.*, we found that in the early stages of the record there was one single globule which passed through the right side of the heart to the pulmonary arteries, thus confirming the subsidiary flow from the inferior vena cava illustrated in Figs. 8A and 8B; Diagram H, p. 687.

SUMMARY

The circulation of the blood through the heart of the adult and the fœtus is demonstrated by the analysis of X-ray cinematographic films recording the passage of injections of radio-opaque media.

In the adult, the blood from both venæ carvæ is seen to pass through the right side

of the heart and the pulmonary arteries to the lungs. On returning from the lungs the flow is seen to pass through the left side of the heart into the systemic circulation.

In the fœtus, the whole of the superior caval blood is seen to pass into the right ventricle and out through the pulmonary valve into the pulmonary trunk; from this vessel it passes on the one hand into the pulmonary arteries and, on the other, *via* the ductus arteriosus, into the descending aorta. As the brachio-cephalic artery leaves the aorta proximal to the entry of the ductus arteriosus, the superior caval blood does not pass to the coronary system, the head and fore-limbs. The inferior caval blood has a double course through the heart. The main part goes through the foramen ovale to the left auricle and ventricle, and passes out into the aorta and coronary and brachio-cephalic arteries. Hence, the heart and brain are given preferential treatment with respect to the supply of oxygenated blood coming from the placenta. On the other hand, a minor part of the inferior caval blood passes with the superior caval flow into the right ventricle and out into the pulmonary arteries and, *via* the ductus arteriosus, into the descending aorta.

ACKNOWLEDGMENTS

Once again we have to express our thanks to Miss M. M. L. Prichard and Mr. M. S. Tuckey for their invaluable assistance throughout the work. In many of the operative procedures a considerable number of assistants have come to our aid, and in particular we wish to mention Messrs. J. Badenoch, J. L. Boldero, J. D. Little, and M. Weatherall. Finally, we wish to acknowledge the grants from the Medical Research Council, which have enabled us to carry out this work.

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EXPERIENCES WITH ENCEPHALOGRAPHY IN CEREBELLAR TUMOR

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THE purpose of this paper is not to encourage the use of encephalography in the diagnosis of tumors of the posterior fossa but merely to describe our experience with spinal air insufflation in these cases. Most authorities do not favor the spinal injection of air in the study of brain tumor suspects, except perhaps in cases of supratentorial lesions with a spinal pressure of 200 mm. of water or less. Its usage in lesions of the posterior fossa is universally and emphatically condemned. In posterior fossa tumors, sudden death from cerebellar hernia has been described after a diagnostic lumbar puncture, making encephalography seem doubly hazardous. Some authors believe that encephalography has no place in the diagnosis of any brain tumor because of danger to the patient, and because the distortion of the ventricles produced by a tumor may prevent the air from entering them. We feel that the simplicity of encephalography, as compared with ventriculography, and the more complete delineation of intracranial structures in the successful encephalogram are potent arguments for its use in suspected supratentorial lesions.

The neurosurgical service of the Cleveland Clinic has used encephalography in patients with suspected cerebral tumor almost regardless of the degree of intracranial pressure. In cases with increased pressure, however, the patient and the operating room are always prepared for craniotomy before proceeding with encephalography. Ventriculography is preferred where the clinical signs favor a posterior fossa tumor. Nevertheless, a survey of the files discloses that encephalography was inadvertently employed in 24 subsequently verified cerebellar cases during the past ten years. In the majority of these cases, of course, the air failed to enter the ventricles but our experience now enables us to make an accurate diagnosis from the distribution of the subarachnoid air.

METHOD

The procedure of encephalography is as follows: If the patient has an increased intracranial pressure, preparations are made for immediate craniotomy. If the pressure is very high, 100 cc. of 50 per cent sucrose is given intravenously. The patient is then anesthetized, usually with avertin, and placed in the encephalogram chair. A lumbar puncture is performed and the needle is connected to a water manometer and to a 10 cc. syringe. After recording the initial pressure, 10 cc. of air is injected, after which the fluid is withdrawn in 5 cc. amounts and substituted with air in similar amounts until no more fluid can be obtained. Roentgenograms are made in the erect posture, and also in the horizontal posture if deemed advisable. The exposures are made, the films developed and read on the operating floor.

A single lateral film of each case taken in the erect position is reproduced in order to illustrate the striking similarity of most of them (Figs. 1 to 5).

ENCEPHALOGRAPHIC FINDINGS

The most common and striking encephalographic finding in these cases is the presence of air over the mesial surfaces of the cerebral hemispheres outlining the supracallosal and cingulate sulci. Of the 24 cases, one or both of these sulci are well visualized in 20 instances. The supracallosal sulcus is more rounded and closer to the cranial vault than in the normal picture. This is due to the upward bulging of the corpus callosum from the obstructive hy-

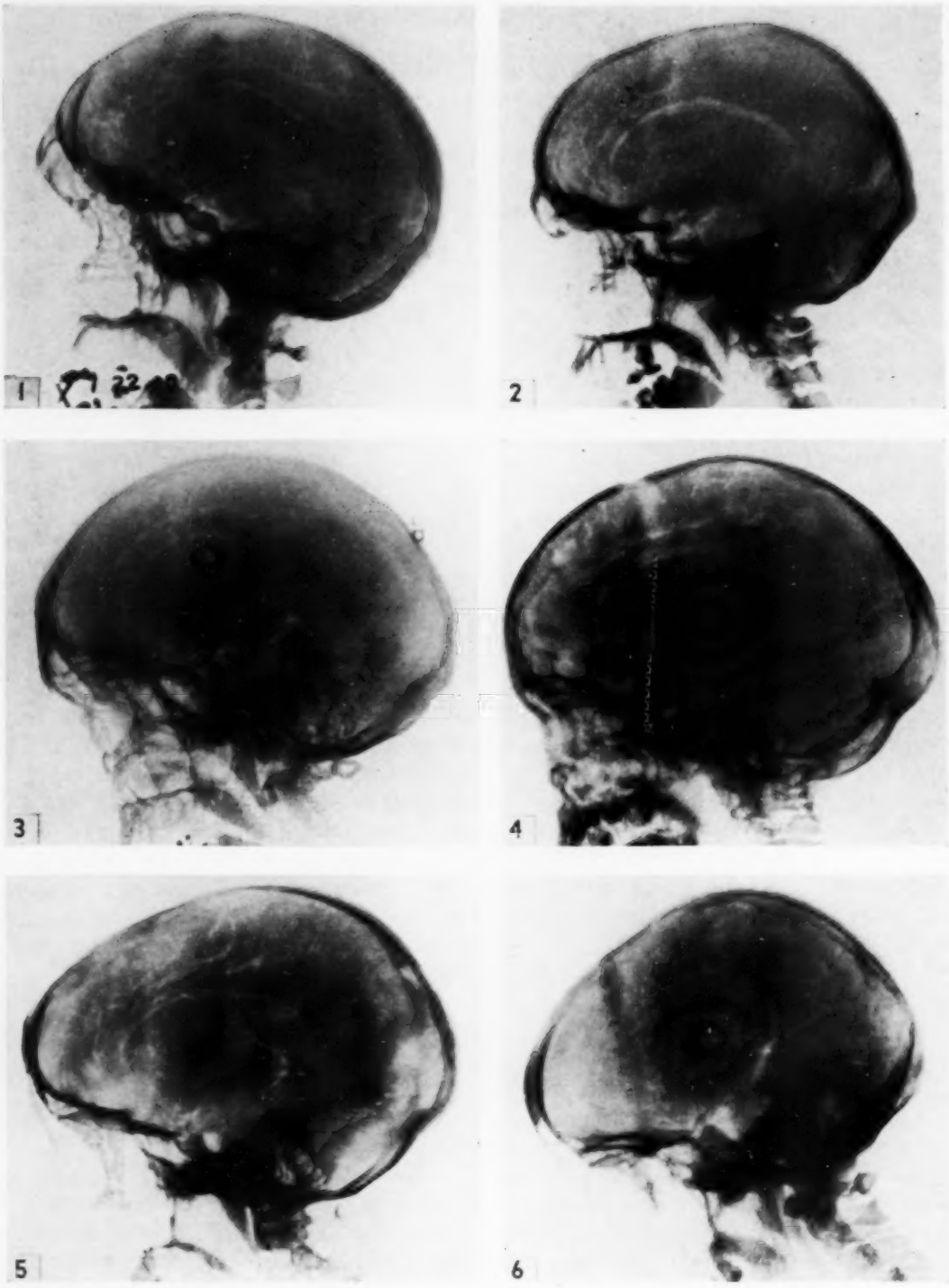

FIG. 1.

Case 1. Arachnoid cyst of left cerebellopontine angle.

Case 2. Cystic hemangioblastoma left cerebellum.

Case 3. Cystic hemangioblastoma left cerebellum.

Case 4. Medulloblastoma of vermis. This patient had bilateral subdural hematomas evacuated on a prior admission.

Case 5. Medulloblastoma of fourth ventricle.

Case 6. Astroblastoma left cerebellum.

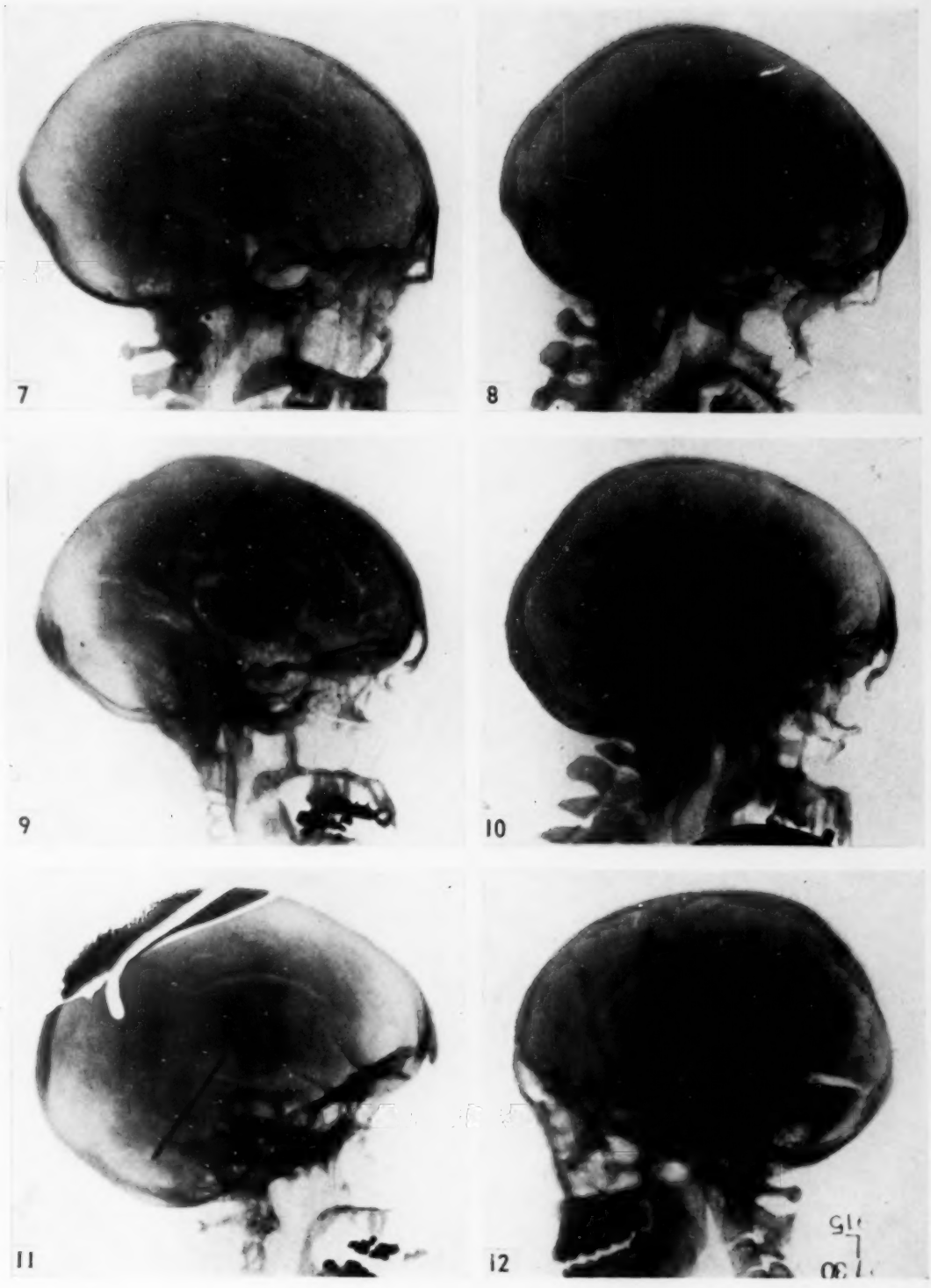

FIG. 2.
Case 7. Medulloblastoma of vermis.
Case 8. Medulloblastoma of vermis. The cisterna magna is largely obliterated by a cerebellar hernia.
Case 9. Metastatic carcinoma left cerebellum.
Case 10. Medulloblastoma of fourth ventricle.
Case 11. Neurofibroma of left eighth nerve.
Case 12. Stricture of aqueduct of Sylvius.

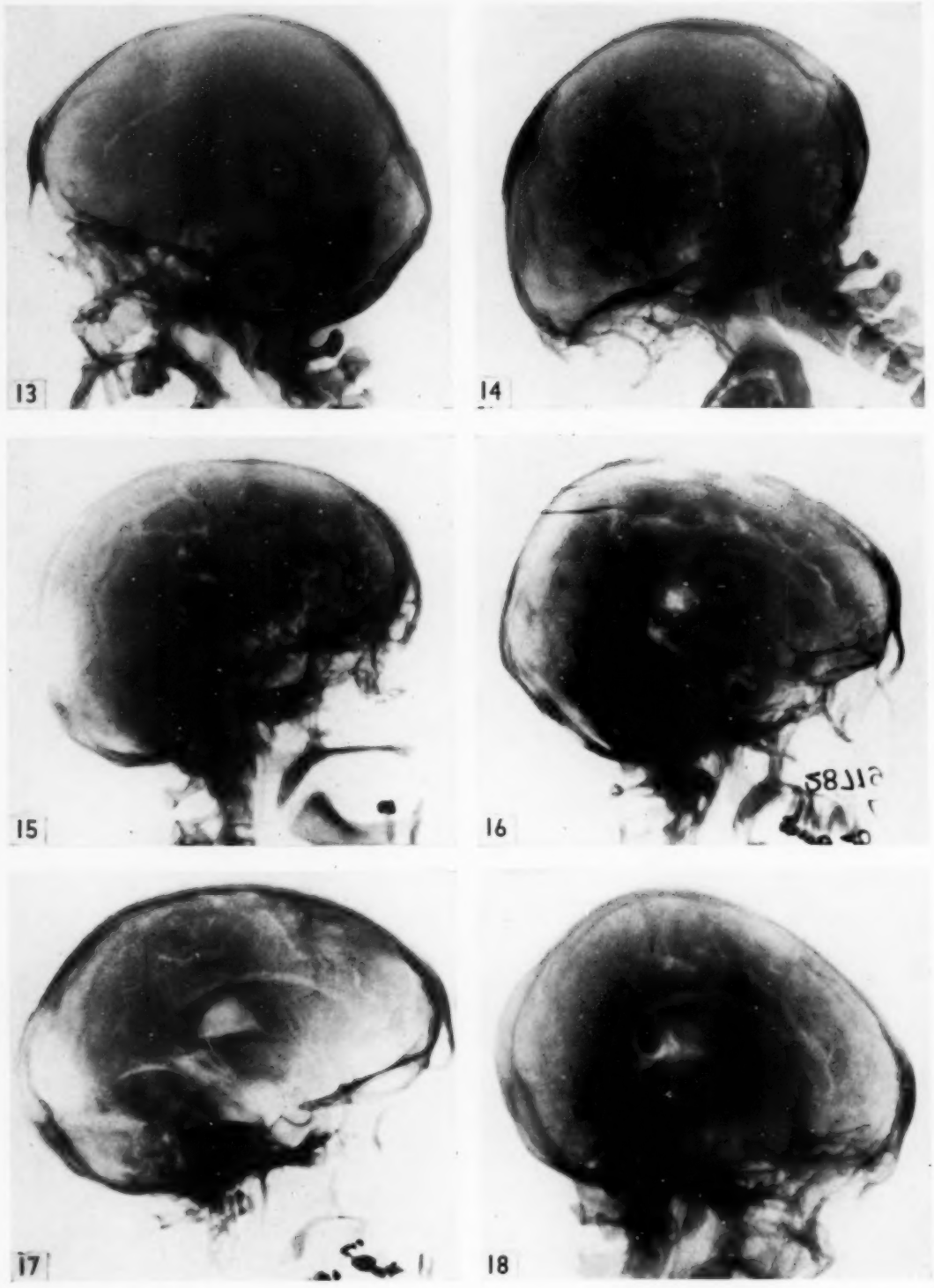

FIG. 3.
Case 13. Cystic astrocytoma left cerebellum.
Case 14. Cystic hemangioblastoma left cerebellum.
Case 15. Metastatic carcinoma. The exaggerated diploic channels partially obscure the subarachnoid air.
Case 16. Cystic hemangioblastoma left cerebellum.
Case 17. Meningioma left cerebellum.
Case 18. Stricture of aqueduct of Sylvius.

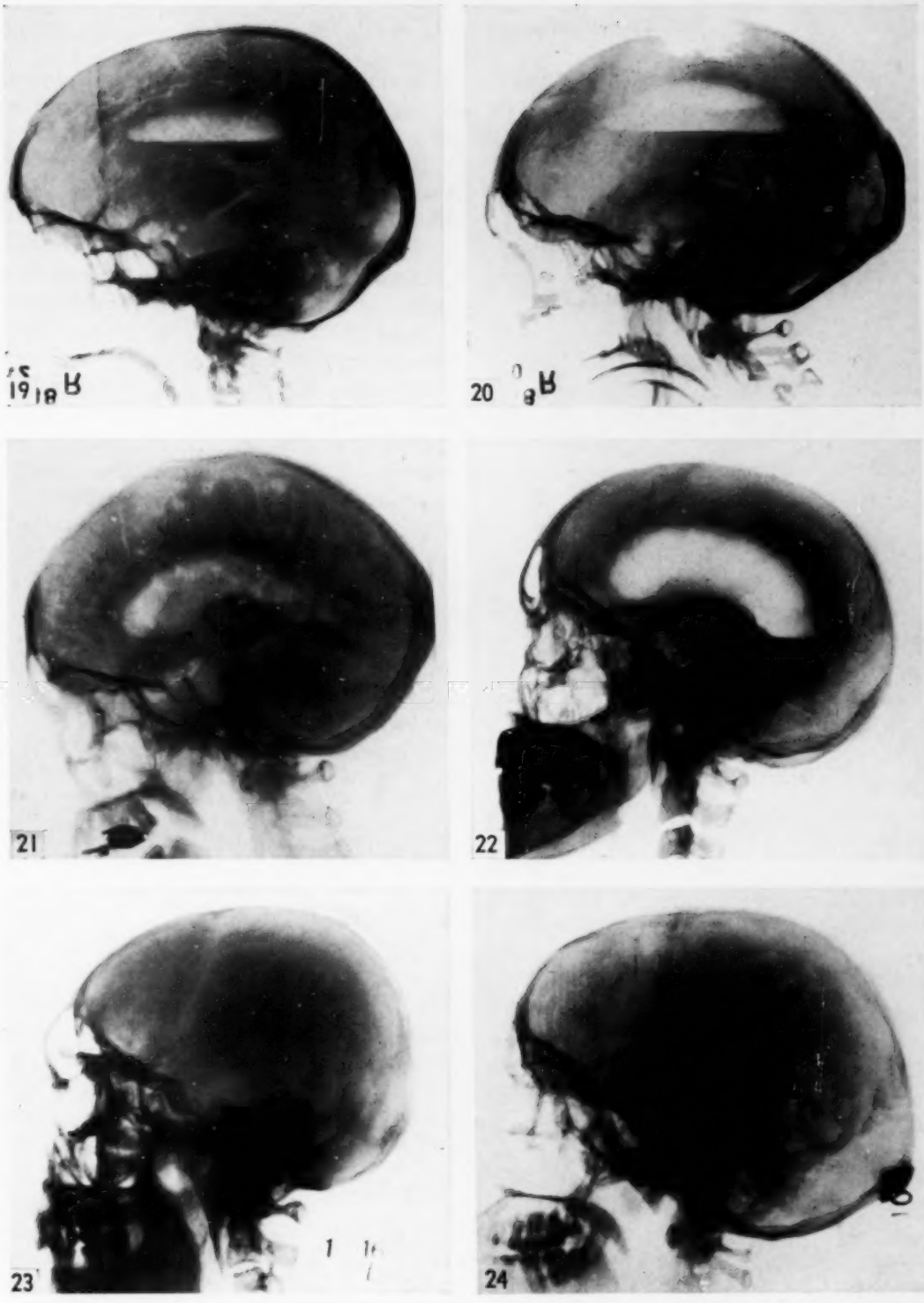

FIG. 4

Case 19. Cystic astrocytoma left cerebellum.
Case 20. Stricture of aqueduct of Sylvius.
Case 21. Medulloblastoma of fourth ventricle.
Case 22. Neurofibroma of left eighth nerve.
Case 23. Arachnoid cysts, bilateral.
Case 24. Cystic astrocytoma right cerebellum.

FIG. 5. Anteroposterior view of Case 1. showing supracallosal air in the midline.

drocephalus beneath it. It is not clear why the subarachnoid air should so selectively seek out the mesial surfaces rather than the convexities of the cerebral hemispheres.

The second most common finding is the presence of a cerebellar hernia. This is shown on the films by a filling defect in the triangular portion of the cisterna magna which lies just below the foramen magnum. In the accompanying illustrations, this is best shown in Case 8. The original films showed a cerebellar hernia in 17 instances. It was doubtlessly present in additional cases but was not visualized because of overexposure of that portion of the film or because of interference of the mastoid process.

In practically all cases the pontine cisterns are flatter than normal as indicated by the proximity of the pons to the clivus. This picture is produced by the crowding of the structures in the posterior fossa by the expanding growth. This finding has been described by Dyke and Davidoff.[1]

Air entered the ventricles in only 4 cases. In 2 of these (Cases 19 and 20), there was a small amount in the lateral ventricles, and in 2 cases (Cases 21 and 22) the lateral and third ventricles were well outlined and presented the usual picture of obstructive hydrocephalus. In no instance could the fourth ventricle be identified definitely. In 2 cases (Cases 23 and 24) no air could be demonstrated within the cranial cavity. In 3 cases (Cases 16, 17 and 18) a curious appearance was produced by an extraventricular collection of air beneath the corpus callosum and just in front of the posterior genu.

The films gave no indication of the exact location of the lesion in the posterior fossae and no hint as to its possible anatomic structure.

SUMMARY OF CASES

The pre-encephalogram diagnoses in these 24 cases were as follows:

	Cases
Brain tumor—unlocalized	10
Brain tumor—possibly cerebellar	10
Probable cerebellar tumor	4

Encephalography was performed in the 4 cases in which the clinical diagnosis was "probable cerebellar tumor" because there was no choking of the optic discs.

The final diagnoses in these 24 cases were as follows:

	Cases
Medulloblastoma cerebelli	6
Cystic hemangioblastoma	4
Cystic astrocytoma	3
Arachnoid cyst	2
Acoustic nerve tumor	2
Metastatic carcinoma	2
Meningioma	1
Astroblastoma	1
Non-neoplastic stricture of aqueduct of Sylvius	3
Total	24

Of these 24 cases the lesions were on the left side in 12 instances, in the midline in 9 instances, and definitely limited to the right cerebellum in only 1 case. The occurrence of a number of midline lesions in this series is

[1] Dyke, C. G., and Davidoff, L. M. Significance of abnormally shaped subarachnoid cisterns as seen in the encephalogram; correlation with clinical cases. AM. J. ROENTGENOL. & RAD. THERAPY, 1934, *32*, 743–756.

to be anticipated since they are notoriously silent as far as localizing signs are concerned and air studies are therefore frequently necessary. But of what significance is the preponderance of left sided lesions as compared to the right? Can it be that right cerebellar lesions are more readily diagnosed clinically because the ataxia involves the dominant hand?

One might well question why air studies were necessary in 2 cases of acoustic tumor since the clinical signs in these cases are usually so clear. The reasons are as follows: One of these patients at the time of her admission was totally blind, psychotic, and uncooperative, and no relatives were available for questioning. Therefore, a satisfactory neurologic study was impossible, and encephalography was resorted to. In the second case, a diagnosis of acoustic nerve tumor was strongly suspected, but the vestibular test showed a hyperactive labyrinthine response on both sides, which cast considerable doubt upon the clinical diagnosis. At operation there was found a rather small acoustic nerve tumor surmounted by a large cyst.

In this series of cases the youngest patient was eleven and the oldest sixty-two years of age. Six patients were in the second decade of life, 5 in the third, 6 in the fourth, 5 in the fifth, 1 in the sixth, and 1 in the seventh.

Sixteen of these patients presented a definite choking of the optic discs; in 4 cases the optic discs were hazy, and in 4 others they were normal. A preliminary spinal puncture was performed in 22 of the 24 cases. In 4 cases the spinal fluid pressure was below 200 mm.; in 7 cases it was between 200 and 290 mm.; in 7 cases it was between 300 and 390 mm.; and in 4 instances it was over 400 mm. of water.

At encephalography all the fluid which could be obtained was removed. The average volume of cerebrospinal fluid recovered in the 24 cases was 73 cc. The largest amount removed in a single case was 195 cc. and the lowest amount was 10 cc. This latter patient had had a spinal puncture three days previously.

In 9 of the 24 cases the encephalogram was followed by a ventriculogram. In the remaining cases, a suboccipital craniotomy was performed on the basis of the clinical and encephalographic findings. A suboccipital craniotomy was performed the same day as the encephalogram in all but 2 of the 24 cases.

POSTOPERATIVE COURSE

Only 3 of these patients showed any immediate deleterious effects from the encephalographic procedure. Case 7 was a young man eighteen years of age whose clinical diagnosis was a brain tumor, possibly of the posterior fossa. His spinal fluid pressure was 400 mm. of water, and there were 4 diopters of edema of the optic discs. At encephalography 75 cc. of fluid was replaced by 85 cc. of air. The resulting films showed good visualization of the supracallosal and cingulate sulci, and a cerebellar hernia could be seen protruding into the cisterna magna. Following this procedure the patient developed respiratory embarrassment which responded to a ventricular tap. A suboccipital craniotomy was performed and a diagnosis of medulloblastoma of the vermis made from the biopsy. This patient did not recover consciousness following operation, although the respirations were satisfactory. The temperature increased rapidly to 104° F., and he died twenty-two hours after operation. Death in this case was attributed to encephalography.

In Case 8 the clinical diagnosis was cerebellar tumor. Although the spinal fluid pressure in the horizontal position was 300 mm. of water, the optic discs were normal. At encephalography the spinal pressure was low and only 10 cc. of fluid could be recovered. This was replaced by 60 cc. of air. The resulting films showed good visualization of the supracallosal and cingulate sulci with the presence of a cerebellar hernia. Following this procedure the patient's pulse became irregular, the face became pallid and sweaty, and the respirations shallow. At operation a medulloblastoma of the left cerebellar lobe was found and

this lobe was amputated. The patient's convalescence from this procedure was satisfactory and he lived three years after operation.

Case 23 was a man, aged thirty-seven, who had a diagnosis of an unlocalized brain tumor. The spinal fluid pressure was 310 mm. of water. There were 4 diopters of choking of the optic discs. At encephalography 55 cc. of fluid was obtained and replaced by 55 cc. of air. The resulting films showed no air within the cranial cavity. During the procedure, which was carried out under local anesthesia, the patient had a generalized clonic convulsion. He recovered satisfactorily from the convulsion and was prepared for a suboccipital craniotomy. During the administration of colonic ether anesthesia respiratory embarrassment developed which responded satisfactorily to a ventricular tap. The operation was continued and an arachnoid cyst of the posterior fossa was evacuated. His subsequent convalescence was satisfactory and he lived for one year after operation.

Two patients who showed no immediate reaction to encephalography died following operation. Their cases were as follows:

Case 6 was a boy seventeen years of age who had a clinical diagnosis of a left cerebellar tumor. His spinal fluid pressure was 210 mm. of water. The ophthalmoscope revealed some fullness of the retinal veins without edema of the discs. At encephalography 58 cc. of fluid was replaced by 75 cc. of air. The encephalogram showed good visualization of the supracallosal and cingulate sulci with no air in the ventricular system. Since these films confirmed the clinical diagnosis, a suboccipital craniotomy was performed and a tumor of the left cerebellar lobe disclosed. Biopsy of this tumor showed it to be an astroblastoma. This patient's convalescence was satisfactory for four days, after which time he developed a hyperthermia which could not be controlled. He died on the twelfth postoperative day. We do not believe that his death was attributable to encephalography.

Case 12 was a young woman, aged nineteen, who had a clinical diagnosis of a probable suprasellar cyst. The spinal fluid pressure was 260 mm. of water. The optic discs showed papilledema with secondary atrophy. At encephalography 70 cc. of fluid was removed and replaced by 60 cc. of air. The resulting film showed good visualization of the supracallosal sulci, and some air in the cingulate sulci but none in the ventricular system. This patient showed no ill effects from the encephalographic procedure. She was not operated upon immediately. Two days after encephalography a ventriculogram was made which showed an enormous hydrocephalus due to an obstruction in the lower end of the aqueduct of Sylvius. A suboccipital craniotomy was performed eight days later and an attempt was made to catheterize the aqueduct of Sylvius. This attempt was not successful and the patient died twelve hours after operation. We do not believe that her death was attributable to encephalography.

The survival period of the 21 patients who were discharged from the hospital varied from two months to seven and a half years. Nine of these patients are living at the time of this report. Therefore, it may be said that of 24 patients with obstructive hydrocephalus due to a lesion of the posterior fossa, there was an unfavorable reaction to encephalography in 3 instances, leading to death in 1 case.

SUMMARY

Encephalography has inadvertently been performed in 24 cases of obstructive hydrocephalus due to posterior fossa lesions. Characteristically the films show absence of air in the ventricles, unusual prominence and curving of the supracallosal and cingulate sulci, flattening of the pontine cisterns, and the presence of a cerebellar hernia. The films do not serve to localize the lesion further than to indicate that it is in the posterior fossae. Of these 24 cases there were unfavorable reactions to encephalography in three instances, leading to death in one case. Ventriculography is preferable to encephalography in the diagnosis of cerebellar tumor.

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THE ELECTROENCEPHALOGRAM AND PNEUMOENCEPHALOGRAM IN NON-FOCAL NEUROLOGICAL DISORDERS*†

By E. H. TROWBRIDGE, JR., M.D., *and* KNOX H. FINLEY, M.D.
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IN THE study of cases of brain tumor and other focal brain diseases, the value of the electroencephalogram and the pneumoencephalogram is sufficiently familiar. In other cases of brain disease, even where there is no symptom of a focal lesion, the electroencephalogram may also be of value. The interpretation of the various abnormal types of electroencephalogram is still a matter of active investigation. Pneumoencephalography is also a method of recognized value in various forms of cerebral disorders quite apart from those with processes which involve either atrophy or displacement of the cerebrum. It seemed worth while to compare the results obtained by these two methods: to see how far the results of the two methods tended to supplement, confirm, or conflict with each other.

The cases observed were patients where there was no suspicion of any focal structural disorder such as tumor, cyst, hemorrhage, or abscess. The one needle pneumoencephalographic technique by way of the lumbar route was used under evipal or pentothal sodium anesthesia. A six channel Grass electroencephalographic apparatus was used to record the brain wave patterns. The pneumoencephalograms were interpreted by the consulting roentgenologists.‡

Records were taken from electrodes placed over the frontal, precentral, and parietal-occipital regions of each hemisphere, with the reference electrode on the mastoid. In several instances recordings were obtained from eight electrode placements over each hemisphere. The ages of patients ranged between nine and sixty-two years. In evaluating the electroencephalographic tracings, the age of the patient was taken into consideration (Gibbs and Gibbs[1]). The electroencephalographic tracings on these patients were divided into three groups: normal, borderline, and abnormal (Fig. 1).

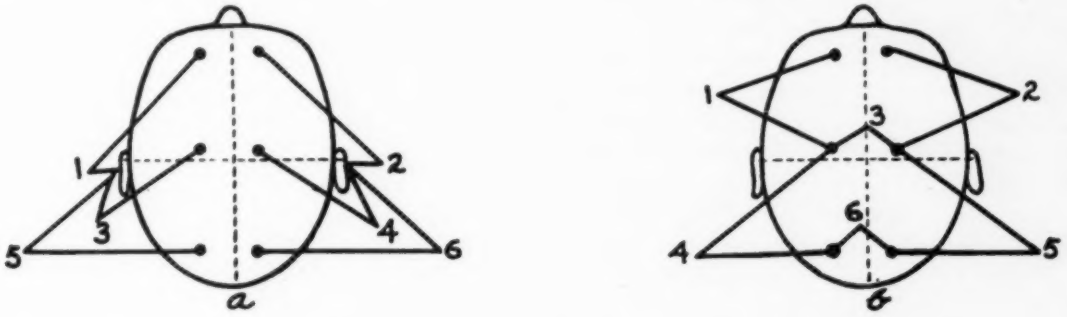

Diagram illustrating the electrode placements of the two types of six simultaneous recordings. *a*, with reference ear electrode; *b*, without reference ear electrode.

OBSERVATION AND DISCUSSION

In 36 of the 68 cases studied, the pneumoencephalogram was abnormal. Of these

‡ We are indebted to Drs. Coffin and Sosman for their interpretation of the pneumoencephalograms.

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† Work supported by grants from the Scottish Rite Masons and Proctor Funds.

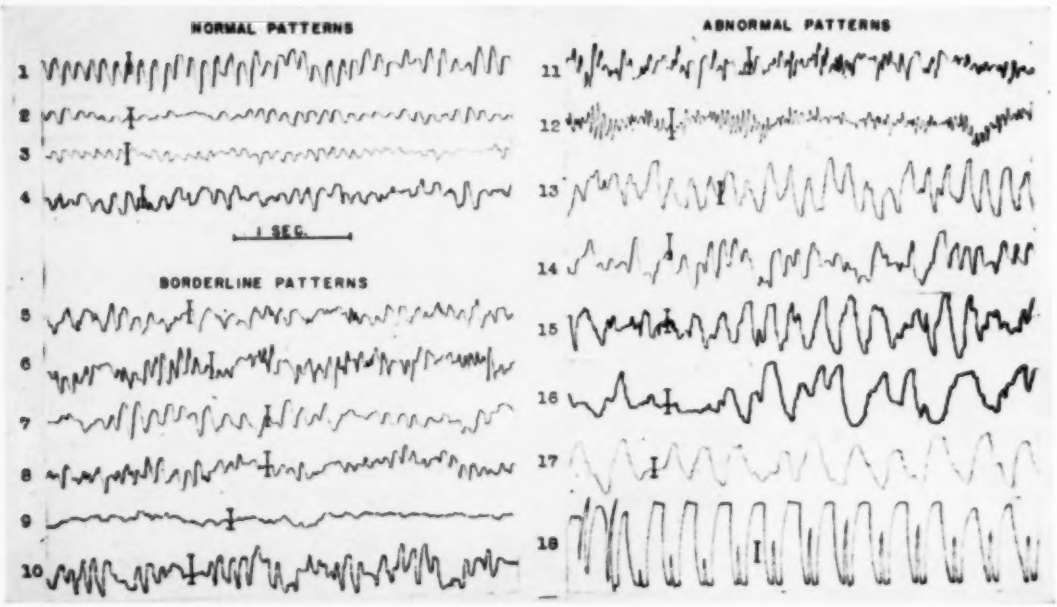

FIG. 1. Samples of normal, borderline, and abnormal patterns. The vertical ink line drawn through the tracing is equivalent to 50 microvolts while the horizontal line represents one second of recording.

36 abnormal cases, 21 revealed cortical atrophy and 15 dilated ventricles. Ten of the latter also had some degree of cortical atrophy. The presence of abnormal pneumoencephalograms seemed to have no direct correlation with the abnormality of the electroencephalographic tracing; for the group of abnormal pneumoencephalograms showed no greater frequency of abnormality of electroencephalogram than the group with normal pneumoencephalograms. In no case with evidence by pneumoencephalogram of dilated ventricles was the electroencephalographic tracing normal. The fact that borderline or abnormal electroencephalograms were always found in cases of dilated ventricles even without cortical atrophy suggests that abnormalities in the basal structures of the cerebrum can modify normal cortical activity. This point was discussed by one of us in a recent study of electroencephalographic tracings in cases of neurosyphilis.[2]

A more detailed analysis of the electroencephalogram revealed 23 patients with tracings showing slow activity (samples 13, 17, Fig. 1), 12 with organized rapid activity (sample 12, Fig. 1), 12 with rapid and slow activity (sample 11, Fig. 1), and 2 with spike and wave activity (sample 18, Fig. 1).

TABLE I

DIFFERENT FREQUENCY PATTERNS IN RELATION TO THE TYPE OF PNEUMOENCEPHALOGRAMS

Electroencephalographic Pattern	Number of Tracings	Pneumoencephalograms			
		Normal	Atrophy	Dilated Ventricles	Dilated Ventricles and Atrophy
Slow	23	8	8	5	2
Rapid	12	7	4	1	0
Choppy	7	2	3	1	1
Mixed	12	8	2	1	1
Wave and spike	2	1	1	0	0

It will be seen from Table 1 that, out of 23 cases showing slow activity, 7 had dilated ventricles, while out of 12 cases showing rapid activity only one showed dilated ventricles. Other than this one differential point, there was found no significant relationship between the cycles frequency of the electroencephalographic and the pneumoencephalographic findings.

For the present, it would appear from these studies that neither the electroencephalogram nor the pneumoencephalogram can be used as a substitute for the other (Fig. 2). Either procedure may give proof of a cerebral disorder without confirmatory evidence from the other. When we come to understand the neurophysiological significance of the different types of electroencephalographic patterns, we may be in a position to predict certain findings in the pneumoencephalogram from electroencephalographic tracings. The fact that in our own series we found no normal records in cases showing dilated ventricles and that rapid activity is uncommon in these cases is a step in this direction.

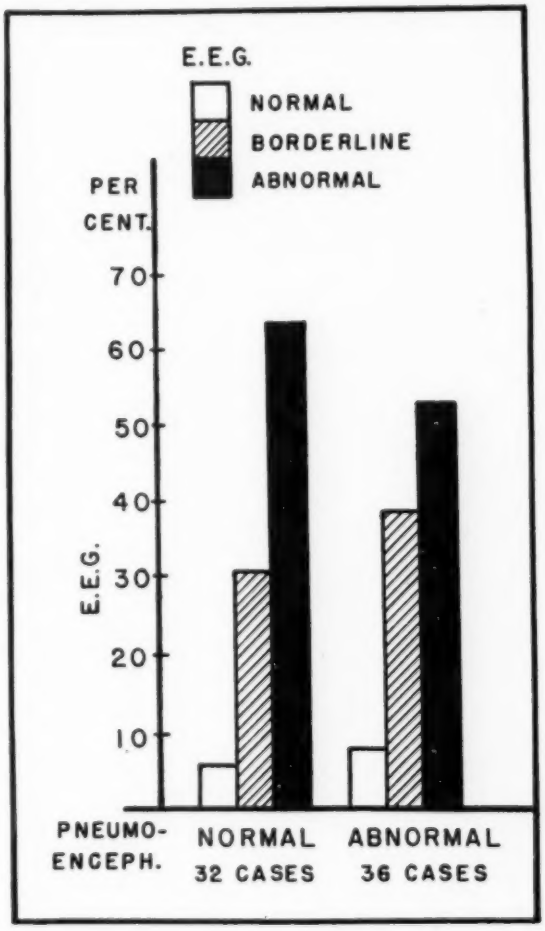

Fig. 2. The percentage of normal, borderline, and abnormal electroencephalograms in cases with normal and abnormal pneumoencephalograms.

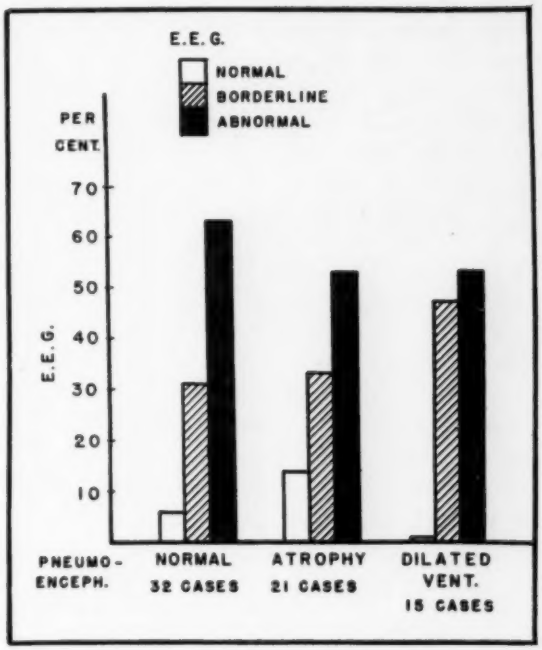

Fig. 3. The percentage of normal, borderline, and abnormal electroencephalograms in normal and different types of abnormal pneumoencephalograms.

SUMMARY

1. A study was made of electroencephalographic tracings and pneumoencephalograms in 68 patients suspected of having a neurological disorder of the non-focal type.

2. In these cases, abnormal electroencephalographic tracings were about as frequent in cases with normal as those with abnormal pneumoencephalograms.

3. Slow activity was common and rapid activity uncommon in cases showing dilated ventricles.

4. Normal electroencephalographic trac-

ings, although found in cases showing only cortical atrophy, were not found in cases with dilated ventricles even without cortical atrophy.

5. The electroencephalogram should be looked upon as adjunct to and not a substitute for the pneumoencephalogram.

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TUMORS OF THE ACOUSTIC NERVE
FROM A ROENTGENOLOGICAL VIEWPOINT

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TUMORS of the nervus acusticus or eighth cranial nerve account for about 8 to 9 per cent of all intracranial new growths. The lesion usually arises from the distal or shorter portion of the nerve which is the portion structurally similar to a peripheral nerve. The proximal or longer portion of the nerve is composed of neuroglia and nervous tissue. These two portions differentiate at about the level of the internal acoustic meatus. Bailey points out that the neoplastic cells of which these tumors are composed resemble strongly the cells forming the neurilemma. He also suggests that the origin of the tumor or its "anlage" may in some way be associated with the tendency to hypertrophy of the neurilemma, a change not infrequently found in and about the ganglion of the vestibular portion of the nerve. Mitotic figures are not commonly found in the cells comprising the acoustic nerve tumors.

A proper term with which to designate these rather odd tumors is not easy to find. Mallory is of the opinion that they should be known as perineural fibroblastomata, and there is much to be said in favor of such a terminology from both a structural and an original point of view. Masson, however, feels that the tumors originate from the cells of Schwann and so should be designated as schwannomata. But as the tendency today is to get away from the name of an individual, in medical terminology, this name has not found wide acceptance. Others have called them peripheral gliomata, but as gliomata are supposed to arise from neuroglia such a name is rather contradictory. Fowler suggested the name neurilemmoma, and such a designation has accuracy to recommend it. Verocay coined the term neurinoma, and neuroma has also been suggested. Fowler feels that these latter terms are misleading because they mean a nerve fiber tumor or nerve tissue tumor, and as the tumors contain no neurites these names are not truly descriptive. Such discussions are chiefly of academic interest, but if we are going to give a tumor a descriptive name it really should be basically accurate. It would seem, then, that the term "neurilemmoma," or the longer name "perineural fibroblastoma," are the two most accurately descriptive terms so far suggested. However, as many believe the tumors are basically of neurogenic origin, the names "neuroma" and "neurinoma" are in wide use.

Tumors of the acoustic nerve, as a rule, develop in patients during their third decade, although no age is immune. Such tumors are not apt to be found in persons beyond the age of fifty, and if one is found in a young person it is not infrequently associated with neurofibromatosis. Most of the cases of bilateral acoustic nerve tumors have been reported as being associated with either a central or a generalized form of neurofibromatosis, and often a familial tendency toward the formation of such lesions can be demonstrated. Wishart was among the first, if not the first, to publish a description of bilateral tumors. His report appeared in 1822 and was quoted by Cushing in 1917. In 1897, Berggrün reported a case of bilateral tumors in an eleven year old child. It is always well to remember that neurofibromas, wherever they may be found, are not always as benign as we are accustomed to believe them to be. Cases have been reported of malignancy being associated with such tumors either through a degenerative metamorphosis of the lesion itself, or possibly by some obscure change being produced in the immediately adjacent tissues.

Acoustic nerve tumors grow very slowly, in fact the symptoms may extend well over

ten years, and as the symptoms are rather insidious in their onset, the tumor may be present for some time before any evidence of its presence will be noticed. It is well known that in the presence of a small tumor there may be no symptomatology whatever, so that we have no accurate method by which we can determine how long an acoustic tumor has been present before the patient seeks medical advice.

The tumor is usually seen as a reddish yellow mass which is fairly well encapsulated, and situated immediately adjacent to the internal auditory meatus and the base of the skull. The tumors may contain regions of degeneration and consequent cysts. But it is rare, indeed, to find calcium in sufficient amount to cast a shadow on the film. Some tumors will simulate meningiomas or astrocytomas. As previously noted, the tumor often arises within the auditory canal near the ganglion of the eighth nerve, so that it will actually be growing out of the auditory meatus and into the cerebellopontile angle. Thus it is that such a growth is often designated as a cerebellopontile angle tumor. This angle is formed by the petrous pyramid, the tentorium cerebelli, the cerebellum and the brain stem. If the anatomical relations of this location can be clearly visualized, the symptoms produced by such a lesion which disturbs this relationship may be easily understood.

Symptoms of an acoustic nerve tumor usually begin with tinnitis which gradually becomes worse, and the hearing will become impaired. Occasionally the symptoms will remain in this stage for long periods of time as the tumor may cease growing. In fact, cases have been reported which have had no loss of hearing. Later, if growth is resumed, the nerves which originate from the brain stem in the cerebellar fossa in the neighborhood of the pons and cerebellar peduncles will become involved due to direct compression. These nerves will be, excluding the eighth which is already involved, the trigeminal or fifth nerve, and the sixth or abducens nerve which will be subject to pressure as it passes over the surface of the pons. The seventh or facial nerve is occasionally involved if the tumor is large, but may escape. The ninth or glossopharyngeal and the tenth or vagus nerve are usually involved to some extent. It is thus easy to understand why the symptomatology referable to the cerebellopontile angle is complicated and extensive, and in addition the patient may show a typical staggering "cerebellar gait." A tumor of the acoustic nerve from the point of view of symptomatology is certainly one of the most clearly defined of all the various forms of brain tumors that we are apt to encounter.

Other changes which are not infrequently found as a result of purely mechanical pressure from the presence of a fairly large tumor will be compression of the sigmoid sinus, and possibly the petrosal sinus may be partially occluded. The symptoms, however, would not be sudden as with a thrombosis, and as the occlusion would be slowly produced, collateral venous drainage would usually be enlarged sufficiently to compensate almost if not completely for the altered condition. Some of the branches of the basilar artery may be involved in this compression and obliteration. There may also be found enlargement of portions of the lateral extensions of the cisterna ambiens. When a tumor reaches a size sufficiently large to produce the changes enumerated above, there will probably be an elevation of intracranial pressure which at times may be great enough to press the cerebellar tonsils into the foramen magnum.

It is well to remember, when considering the differential diagnosis of tumors in the cerebellopontile angle, that whereas all tumors in this location will involve the same group of nerves to a greater or lesser extent, the acoustic nerve tumor is the only one which will involve the eighth nerve first. And in addition, tumors in the cerebellopontile angle, other than those of the eighth nerve, are very apt to give rise to increased intracranial pressure relatively early in their course. This emphasizes the importance of a careful history, and the

importance of the clinical findings as a prerequisite to a careful and complete evaluation of the roentgen examination.

If we fail to obtain a clear cut history which would strongly suggest the presence of an acoustic nerve tumor then we must consider in our differential diagnosis several other conditions. The tumors most apt to be found in the cerebellopontile angle would be those of meningeal origin, the meningiomas, those of blood vessel origin, including the vascular anomalies, and tuors which might originate from the parenchyma of the cerebellum, the cerebrum or the pons, chiefly the gliomas. Of these, the astrocytomas or oligodendrogliomas would be the most confusing because their growth is slow. A neuroblastoma or teratoma might at times occur in this location and prove confusing. A papilloma of the choroid plexus at the foramen of Luschka might produce similar symptoms. Sarcomas and other forms of metastatic malignancies might be found but their rapid progress would rarely make one confuse them with the slow-growing tumors under consideration. Other conditions besides tumors must be ruled out when an "angle" lesion is suspected. Such conditions are multiple sclerosis, adhesive arachnoiditis, and syphilitic or tuberculous processes.

The acoustic nerve tumors, like other tumors of the cerebellopontile angle, usually will cause an increase in the protein content of the cerebrospinal fluid. Several cases have been reported, however, in which the symptoms have been aggravated by a spinal puncture, so that this diagnostic procedure must be approached with caution if such a lesion is thought to be present.

Fig. 1. Tumor of the eighth nerve. There is atrophy of the sella turcica, although the anterior clinoids are practically intact.

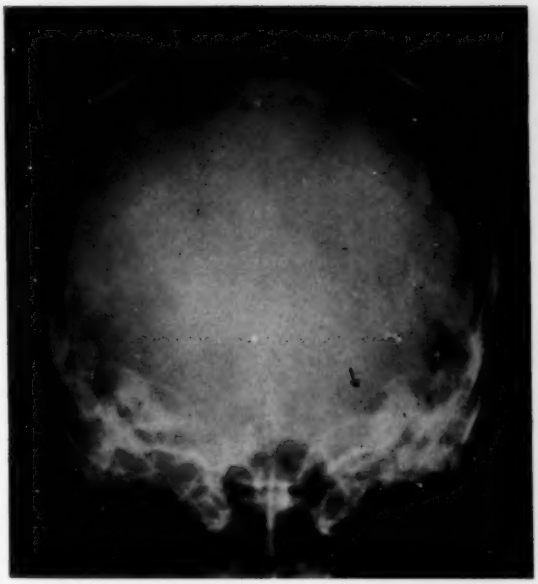

Fig. 2. Same case as Figure 1. Anteroposterior view, showing erosion (arrow) of the left petrous ridge. This is the most typical roentgenographic evidence of a tumor of the eighth nerve.

With the foregoing information as a background, we are now in a position to consider the roentgen examination of a patient suspected of having an acoustic nerve tumor. First of all, the examination must be complete, and carefully correlated with the history and clinical findings, if a lesion in its early stages of development is to be discovered. It might be pertinent to mention here the oft-discovered statement in the literature that "the roentgen examination was negative." This statement, when appearing alone, must be taken with the proverbial "grain of salt." For until we know who made the examination and how complete it was, we are not justified in accepting such a statement at its face value. We are all well aware of the fact that many conclusions are drawn from most inadequate examinations.

To examine adequately a skull suspected of harboring a tumor of the eighth nerve, we must have stereoscopic films of both sides of the head, and films taken in the occipital position, in the semilateral (Stenvers) position, in the nose-chin (Waters) and the brow-nose positions. The latter should be taken with the tube centered between the occiput and vertex and tilted caudad about 5 degrees in order to cast the shadow of the petrous pyramids behind the orbits. If the sagittally taken films are amply exposed the petrous pyramids will be sufficiently penetrated to permit seeing and studying the entire bony portion of the auditory canal. In addition to these positions, it might be well to attempt to obtain films of the internal auditory meatuses, but this is usually not very successful. A better scheme is to use planigraphy when possible as it will show the meatuses to much better advantage.

If the petrosa are symmetrical we can compare the two sides in detail, but unfortunately they are usually asymmetrical so that each must be studied individually. As has been noted, the tumor usually originates in the canal close to the internal auditory meatus, and usually tends to grow through the meatus, although growth in both directions can occur. The walls of the canal may show slight atrophy which would be the earliest roentgen evidence we could expect to find. This atrophy must be real. Slight enlargement or asymmetry does not necessarily indicate atrophy. The cortex must show demineralization to be atrophic, and this can be determined only by comparison with comparable portions of the same bone, or with the bone of the opposite side. If the pyramid is dense and sclerotic, the recognition of early atrophy of the canal is well nigh impossible, but if mastoid cells appear about the canal the problem becomes less difficult. The next step in the development of the tumor would be recognized by an enlargement of the internal auditory meatus with atrophy of its walls. A comparison with the opposite side will usually help in recognizing this change, but if bilateral tumors are indicated by the history we must then rely mostly upon our being able to recognize atrophy *per se*, unless the enlargement is perfectly obvious. It is at this stage that the planigram will be of greatest aid.

The next stage will be when the tumor has emerged from the canal and has produced a notch-like defect in the petrous ridge, usually as part of the destructive enlargement of the internal auditory meatus. The margins of this defect will be definitely atrophic. The further development of the tumor in the cerebellopontile angle before the onset of increased intracranial pressure is difficult to follow roentgenographically. However, an encephalogram at this time may show a distortion, obliteration, or even a cystic enlargement of the homolateral portion of the cisterna ambiens, and also a distortion of the aqueduct. There may also be sufficient transmitted pressure to produce a slight homolateral atrophy of the dorsum sellae, particularly if this structure happens to be of the type most susceptible to atrophy. Here again, the determination of the presence of atrophy must depend upon the careful study of the appearance of the cortex. So often demineralization of the sella turcica is part of a systemic process and is erroneously considered to be due to increased intracranial pressure. A study of the cortex and surrounding bone will usually eliminate such an error. The last stage when the tumor is large would be characterized by all the changes previously mentioned plus evidences of increased intracranial pressure. These evidences have been noted so many times in previous articles that it would be repetitious to mention them again. However, it might be well to warn against considering all cases showing some increase in the convolutional impressions as being due to increased intracranial pressure. The cortex of the interconvolutional ridges and the bone covering the ridges must be really atrophic to be due to pressure. Otherwise the convolutional markings may well be atypical normal variations, or the result of some metabolic dis-

turbance which was a factor in early life.

The pineal gland may be displaced by an acoustic nerve tumor, but such a displacement is much more commonly found in association with a supratentorial tumor. The displacement may be forward and upward if the tumor grows slightly cephalad rather than into the cerebellopontile angle in a caudad direction. This may also produce a slight lateral shift. When the tumor reaches a size sufficiently large to cause a general rise in intracranial pressure, the third ventricle will become dilated and the pineal body may be displaced posteriorly.

We must bear in mind the fact that an actual displacement of the pineal body in the sagittal plane is not as easily determined as we are led to believe. Charts and various measuring devices are available which determine whether or not a given pineal body lies within the "normal zone." These "normal zones" are relatively large, and consequently even if a given pineal gland be found within them, we have no way of telling whether or not it may have been displaced from some portion of the zone to another. The leeway is altogether too great for accuracy in any individual case; however, the determination of a lateral shift can be depended upon to a much greater extent than can the determination of a slight shift in the sagittal plane. If the glomus of the choroid plexus, either singly or bilaterally, contains enough calcium to be seen in the film, then its relationship to the pineal shadow can be estimated in stereoscopic films. If the relationship is evidently normal then, even if the pineal body is apparently displaced by measurements, it is likely that the cause will be found to be anatomical and not pathological. Also, if the relationship between the pineal body and the calcium in the choroid glomuses appears abnormal, even though the pineal be within the "normal zone" it should be suspected as being abnormal. It would be rare, indeed, to find the pineal body and both choroid glomuses displaced in such a manner as not to alter their relative positions. A careful study of the relationship between these structures, whenever they can all be localized, will frequently forestall errors in interpretation both of commission and omission.

This, then, is the way a typical tumor of the acoustic nerve would be expected to develop, and by a study of the films we may be able to tell about what stage the tumor is in at the time of the examination. But, unfortunately, not all such tumors run true to form. Not infrequently we will find a tumor which produces little or no bony change until the state of increased intracranial pressure is reached. In such a case a relatively early diagnosis would have to depend upon the symptoms and clinical findings plus encephalography, which would be expected to show the usual distortions of the cerebral fluid spaces produced by a tumor in the cerebellopontile angle.

Let us now consider in greater detail the differential diagnosis between acoustic nerve tumors and conditions with which they might be confused. First of all, we must never lose sight of the fact that multiple lesions can occur of the same and dissimilar types. Most of us are prone to ascribe all changes noted in an examination to a single cause, particularly if a specific type of lesion is obviously present. In the majority of cases this will be correct, but a careful study of all the variations from the normal seen in any given case will reward us occasionally with the information that more than one disease process is present.

A cancer of whatever origin, invading a petrous pyramid, might produce a notch-like defect closely simulating the change usually associated with a tumor of the eighth nerve. The surrounding bone, however, would have an "invaded" look with indefinite outlines to the defect. The acoustic nerve tumor will erode bone as a result of pressure only. Also the history of such tumors would be entirely different. The cancer would be rapidly malignant.

A primary epidermoidoma of a petrous pyramid occasionally produces a bone defect closely simulating an eighth nerve

tumor still within the canal. This may prove very confusing; however, the bone defect due to an epidermoidoma is apt to be considerably larger than the symptoms would warrant if it were due to an acoustic nerve tumor. And, in addition, if the spinal fluid were examined and found to have an increased protein content, the chances of its being caused by an epidermoidoma would be relatively slight.

Another tumor which occasionally produces notch-like defects of the petrous ridge is a meningioma, usually originating from the walls of the petrosal sinuses. Although the bone defect might be confusing, the symptoms would not be. With a meningioma the eighth nerve would not be involved before other symptoms would be noted. Not infrequently a meningioma in this region will give rise to some bone hypertrophy which would immediately rule out the possibility of its being caused by an acoustic nerve tumor. A meningioma in this location will frequently displace the pineal gland toward the opposite side and possibly somewhat anteriorly. It might be interesting to mention the fact that meningiomas, when found below the tentorium, will not infrequently be associated with neurofibromatosis.

A glioma of the astrocytic or oligodendral types might cause a slight notching of a petrous ridge by direct pressure. Such a contingency would be remote but on rare occasions gliomas do produce bone atrophy by direct or almost direct contact. Such a change would rarely be confusing except as a complicating factor in the presence of two lesions. The glioma would almost certainly displace the pineal body and the signs and symptoms would indicate a lateralized cerebral lesion. If the glioma were actually within the cerebellopontile angle we would not expect any bony change involving a petrous ridge. The gliomas are also apt to calcify and so at least give us a clue to their identity.

Other forms of expanding lesions in or near the cerebellopontile angle would hardly be expected to produce bony changes which might be confusing. However, if our search failed to reveal any suggestively characteristic changes and we found it necessary to resort to encephalography, then if a tumor were present we would find evidences of a lateralized subtentorial lesion, and in the presence of a rather typical history and clinical picture, we would be able to make a fairly definite diagnosis of an acoustic nerve tumor.

After a diagnosis has been made of the presence of an eighth nerve tumor, the question of therapy immediately arises. The treatment of these tumors is primarily surgical, and a complete removal of the tumor will produce a cure. However, not infrequently a complete removal will be impossible or for some reason inadvisable. Under these circumstances radiotherapy in large doses might be employed in the hope of inhibiting the almost inevitable growth of any remaining tumor tissue. These tumors contain very few mitotic figures and are therefore not radiosensitive. For this reason radiotherapy has little to offer as a primary form of treatment.

SUMMARY

The acoustic nerve tumors constitute about 9 per cent of all intracranial new growths; they are slow growing, involve the eighth nerve first, produce atrophy in and about the internal auditory meatus, and when bilateral they are apt to be associated with neurofibromatosis; when large, they invade the cerebellopontile angle and give rise to increased intracranial pressure. The roentgen examination must be complete. It often shows characteristic changes which make a definite diagnosis possible. The treatment of these tumors is primarily surgical.*

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PRIMARY TUMORS OF THE DIAPHRAGM ROENTGENOLOGICALLY CONSIDERED

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THERE are few reports in the literature on diaphragmatic tumors. This is obviously due to the fact that primary neoplasms of the diaphragm are very rare. Since Binney's review of this subject, isolated cases have been reported by Burvill-Holmes and Brody, Müller, Kirshbaum, Söderlund, and Peery and Smith.

All benign and malignant neoplasms of the diaphragm are of mesenchymatous origin. Among the benign tumors of the diaphragm, lipomas, fibromas and an angiofibroma have been reported. They grow slowly and do not have a tendency to undergo sarcomatous transformation. Some of these tumors have been found incidentally on postmortem examinations, without having caused any clinical symptoms.

The malignant tumors of the diaphragm comprise various forms of sarcoma. Fibrosarcoma, fibromyosarcoma, rhabdomyosarcoma, and leiomyosarcoma have been described in the literature. The rhabdo- and leiomyosarcomas have greatly attracted the attention of pathologists because of their rarity. Particularly rare are myogenic sarcomas derived from striated muscle fibers. Undoubtedly very undifferentiated myosarcomas may not be identified in all cases, unless special staining methods are used. The origin of the highly undifferentiated rhabdomyosarcomas was sought in isolated muscle fibers, which have failed to differentiate fully during fetal development. The fact that in a myogenic tumor cell the tendency to differentiation remains rudimentary probably accounts for the failure of cross striations to develop. Sarcomas of the diaphragm grow very rapidly; they often assume large dimensions and displace the lung and mediastinum. Metastases of the lung, pleura, and bones occur very frequently. Pleural metastases are usually associated with a hemorrhagic pleural effusion, which may obscure the true origin of the malignant tumor. Age does not seem to be an important factor in the occurrence of diaphragmatic sarcomas; they have been reported between fourteen and sixty-five years of age. In all cases reported in the literature death resulted within a few months after the onset of clinical symptoms.

The clinical manifestations of diaphragmatic tumors are not very characteristic. However, some of the complaints recurred in most of the cases, and therefore may be briefly summarized: Usually there was a more or less severe pain in the lower chest, referred to the hypochondrium; the pain increased on respiration and occasionally radiated along the distribution of the intercostal nerves. Frequently there was shortness of breath, in some instances the dyspnea was of an obstructive type. Usually there was dry cough; in one case hemoptysis occurred. As the tumors increased in size, bulging of the hypochondrium was observed. In several instances the tumor was palpable in the hypochondrium and moved definitely with respiration. For this reason, enlargement of the liver or spleen has been erroneously assumed in some cases. In a great majority of cases a correct diagnosis of diaphragmatic tumor cannot be expected without the aid of supplementary methods. Of the greatest diagnostic value are thoracoscopy and roentgenography. By thoracoscopy a direct visualization of the tumor and its relation to the surrounding structures may be obtained. However, it is usually preceded by roentgen examination.

If roentgen diagnosis of diaphragmatic tumor is to become efficient, it must be conducted carefully and in a logical sequence. Since tumors of the diaphragm must be differentiated from other intra-

thoracic diseases, and from some abdominal tumefactions which may bear a roentgenological resemblance to pathological conditions of the diaphragmatic pleura or diaphragm, the roentgen examination should consist of several procedures to secure satisfactory diagnostic criteria. These are the procedures in the preferred sequence: (1) roentgenoscopy supplemented by roentgenograms; (2) pneumoperitoneum, and (3) pneumothorax.

(1) Roentgenoscopic observation of the diaphragm on normal respiration and on application of Mueller's test is indispensable for the determination as to whether the tumor is of intrathoracic or abdominal origin. Basal intrathoracic tumors show respiratory motion, synchronous with the normal excursion of the diaphragm. If the tumor lies underneath the diaphragm, and protrudes through it into the thoracic cavity, a paradoxical movement of the tumor shadow is noted, similar to that characteristic of diaphragmatic hernias. The paradoxical movement may not be demonstrable in the presence of adhesions within the diaphragmatic defect. In those cases the tumor mass moves together with the adherent diaphragm. The paradoxical movement, in the absence of adhesions, is due to increased intra-abdominal pressure during inspiration, which presses the abdominal contents upward.

(2) A circumscribed subphrenic mass can clearly be differentiated from a tumor extending into the thoracic cavity by a pneumoperitoneum. A subphrenic tumor is then usually separated from the leaf of the diaphragm by a layer of air, whereas a tumor protruding into the thorax usually remains above the diaphragm. Should such a tumor be reduced (analogous with a hernia) into the abdominal cavity, then the gas escaping through the defect within the diaphragm would produce a pneumothorax, which of course would prove the presence of a diaphragmatic defect.

These signs have been occasionally observed in cases of echinococcus of the liver, protruding into the thorax or in cases of diaphragmatic hernia of the liver, which is not an extremely rare occurrence.

(3) After the intrathoracic origin of a basal tumor has been established, the exact recognition of its relation to the thoracic viscera is facilitated by artificial pneumothorax. This will usually determine whether a shadow noted at the base is of pulmonary or extrapulmonary origin. The intrapulmonary tumors, especially those which do not affect the visceral or parietal pleura, will usually separate from the diaphragm when a good compression of the lung is obtained. A tumor of the diaphragm or diaphragmatic pleura will rest with its base on the diaphragm and be separated from the collapsed lung, which shows no pathological changes. A diagnostic pneumothorax may also solve the question as to whether a shadow resting on the diaphragm is produced by the presence of fluid or by a solid tumor. If, after aspiration of fluid, air is injected and films are taken in different positions, the air will shift with the shifting of the fluid, and so establish the diagnosis of an encapsulated effusion. Air in an otherwise solid tumor will not change its position with change of posture.

If a satisfactory collapse of the lung is prevented by simultaneous involvement of the pleura, the differentiation of the diaphragmatic tumor from pulmonary disease may become impossible. Such a difficulty may be encountered in differentiation between neoplasm of the diaphragm and pulmonary sarcoma. Pulmonary sarcoma may grow either by infiltration and destruction of the pulmonary parenchyma, or by expansion. The expansive growth seems to be more frequent. The growing tumor does not spread along the peribronchial and perivascular channels, as in case of bronchogenic carcinoma. This tendency of pulmonary sarcoma to grow expansively finds its roentgenological expression in the convexity and sharpness of the contours of the tumor. The destructive component of these tumors does not change its contours, but causes a slight loss of sharpness of the shadow.

An echinococcus cyst, which is often found at the base of the lung, shows quite frequently a calcified ring and may then be recognized with relative ease.

More difficult is the differentiation of diaphragmatic tumors from tumors of the pleura. Of these the primary pleural sarcomas must be considered. Macroscopically primary sarcomas of the pleura can be divided into: (*a*) diffuse sarcomatous infiltrations; (*b*) circumscribed, destructively growing sarcoma, and (*c*) expansively growing fibrosarcoma. Only the last two forms of pleural sarcoma deserve careful attention.

The circumscribed, destructively growing sarcomas may assume large dimensions and lead to infiltration of the lung, on the one hand, and on the other, produce marked destruction of the ribs and invade the soft tissues of the thoracic wall. Pleural effusion may be absent. They metastasize to the lung, liver and skeleton.

The expansive fibrosarcoma, of which only a few cases have been reported, presents a peculiar form. It grows entirely expansively, assuming a large size, and occupies a large portion of the lung field. This tumor is loosely attached to the surrounding tissues by thin vascular strands. Otherwise it is fully movable in the pleural cavity. The diaphragmatic pleura is the most frequent origin of this tumor. It never invades the lung. There have never been any retrogressive changes noticed within the tumor. Also the pleural effusion which frequently accompanies the malignant tumors of the pleura is not encountered; it does not produce any metastases. Clinically it causes dyspnea, cyanosis, venectases of the thoracic wall, dullness to percussion, absence of respiratory sounds. The prognosis is favorable because it can easily be removed and does not recur. An encapsulated pleural effusion simulating a neoplasm of the diaphragm can be ruled out only after thoracocentesis and introduction of air, which will establish the diagnosis definitely.

At the University Hospital, we have had an opportunity to study 2 cases in which a diagnosis of a diaphragmatic tumor was made. In one case this diagnosis was confirmed at operation.

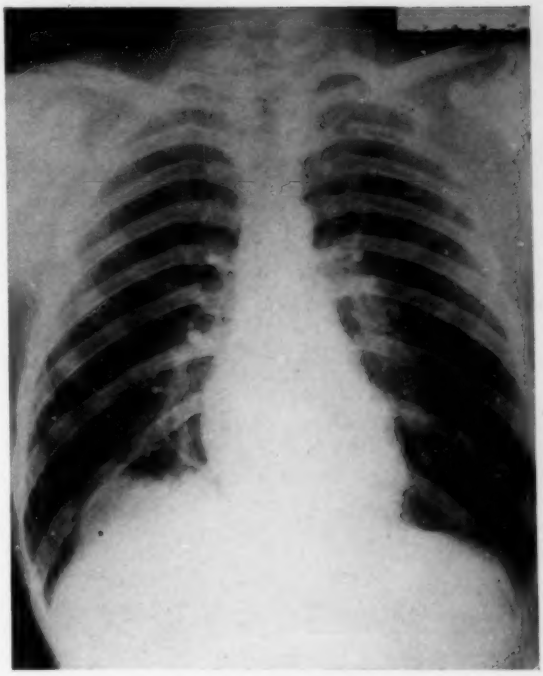

Fig. 1. Case 1. A sharply defined shadow at the right base resting on the diaphragm dome. The upper contour is polycyclic.

Report of Cases

Case 1. F. C., white male, aged twenty-seven, was admitted to the University Hospital in May, 1938. In December, 1937, the patient was seized with severe pain in the right lower chest, increasing on respiration. At first this pain was associated with dry, hacking cough, which later became productive of a thick mucoid material. Later there was marked shortness of breath on exertion, and the patient noticed that the sputum was tinged with blood. The patient was hospitalized elsewhere. A roentgenogram of the chest at that time showed a mass in the right lower lung field. Thoracocentesis was performed and fluid tinged with blood was aspirated. After that his condition improved slightly. In April shortness of breath became worse. The thoracocentesis was repeated and 600 cc. of bloody fluid removed. This gave considerable relief. There was no edema, cyanosis, chills or fever. Patient lost approximately 40 pounds since the onset of his illness.

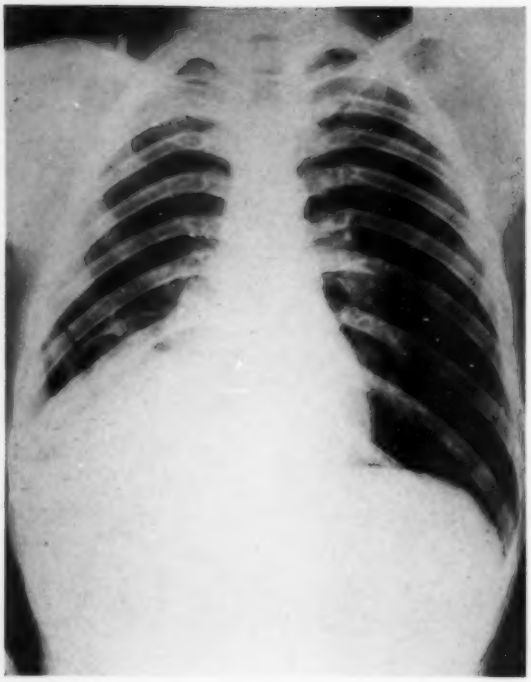

Fig. 2. Case 1. The tumor has increased in size. The cardio- and costophrenic angles are obliterated. Plate-like atelectasis of the lung above the tumor.

On physical examination there was a limited expansion of the right lower thorax, complete dullness to percussion, and on auscultation the breath sounds over the right chest, with exception of the apex, were absent. There were numerous râles in the right apex. The cardiac dullness extended beyond the left mammillary line. The heart tones were normal. The temperature and pulse were normal. Erythrocyte count, 4,875,000; leukocyte count, 7,100, with a normal differential count. Thoracocentesis was repeated shortly after admission to the hospital, and 1,900 cc. of bloody fluid removed. Microscopic examination of the smear showed homogeneous, pink staining fibrinous material, in which there were a few scattered red cells. No malignant cells could be demonstrated. A bronchoscopic examination failed to demonstrate any evidence of a pathological condition of the investigated middle and lower lobe bronchi.

Roentgenograms of the chest were made and compared with those taken elsewhere shortly after the onset of the patient's complaints. The first roentgenogram showed a fairly large, sharply defined shadow of homogeneous density in the right lower lung field, resting with a broad base on the dome of the diaphragm, from which it was not separated by interposed normal pulmonary parenchyma. The superior contour of this shadow presented a polycyclic appearance, suggesting either a lobulated tumor or a mass of multicentric origin. The right costophrenic angle and mediastinum were free; the heart was in midline.

The second roentgenogram, taken approximately one month later, showed an increase in the size of the tumor. The upper contour, still sharply defined, was convex and formed one arc; the right costo- and cardiophrenic angles were obliterated. Above this shadow there was a convex line of increased density in the right lower lung field; the course of this linear shadow followed closely the upper contour of the tumor.

The roentgenogram of chest, after the patient had been admitted to the University Hospital, showed a very large shadow of homogeneous density occupying the lower two-thirds of the right lung field, obliterating completely the right costophrenic angle, and extending laterally to the chest wall. The heart was displaced considerably to the left. The upper, sharply defined contour of the tumor remained convex. On roentgenoscopic examination an anterior and lateral displacement of the esophagus was dem-

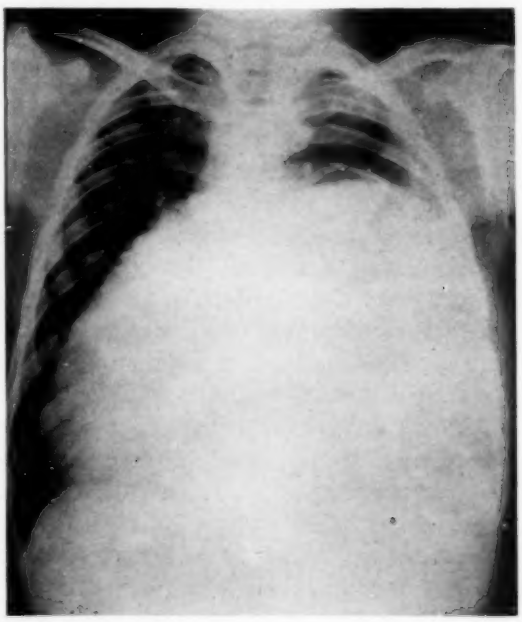

Fig. 3. Case 1. The tumor has grown rapidly. Its superior contour remains convex. The heart and mediastinum are displaced.

onstrated. A diagnostic pneumothorax revealed evidence of fluid at the right base. A pneumoperitoneum showed free air underneath the right diaphragm on roentgenoscopic examination. A diagnosis of a sarcoma of the right diaphragm or diaphragmatic pleura was made and thoracotomy advised.

In July, 1938, the patient was operated upon. A long incision was made in the eighth costal interspace, extending from the axillary line forward to within 8 cm. of the costochondral junction. A 1 cm. section was removed from the eighth rib posteriorly to secure better exposure. A large portion of the tumor was removed by blunt dissection with the fingers from the dome of the diaphragm. Hemostasis was secured. The cavity was closed by interrupted sutures. Partial expansion of the lung was obtained before the final suture was closed. On close inspection the large fungating, friable tumor mass was found to arise from the anterior dome of the right diaphragm. The pleura was adherent in several places. There were metastases in the right lower lobe. About 1,500 cc. of blood-tinged fluid was removed from the pleural cavity by suction.

The pathological examination of the removed tumor showed it to consist of soft, friable, pale yellowish-gray tissue, approximately 8×5×5 cm. The tumor surface was shaggy and extremely irregular with numerous strands of dark gray, necrotic appearing tissue. The cut surface was moist, bulging, and scattered throughout were numerous arborizing trabeculations between which there were numerous small, irregular cavities, filled with semifluid material. In some areas there were irregular cystic cavities varying from 5 to 10 mm. in diameter. Microscopically several sections from different portions of the tumor showed predominantly necrotic tissue. In some of the sections there were small, scattered areas of tumor composed of very embryonic spindle cells without any definite arrangement. There was a tendency toward a radial arrangement of the cells around blood vessels. The tumor cells varied greatly in size and shape. The nuclei were particularly large in many of the cells. There were many mitotic and hyperchromatic nuclei. The stroma was scanty. The histopathological diagnosis was: very malignant type of fibrosarcoma.

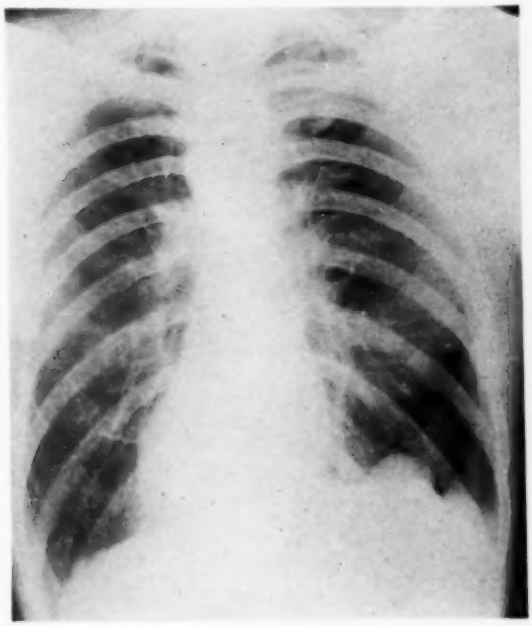

Fig. 4. Case 11. A round, sharply defined shadow at the right base, resting on the diaphragm dome.

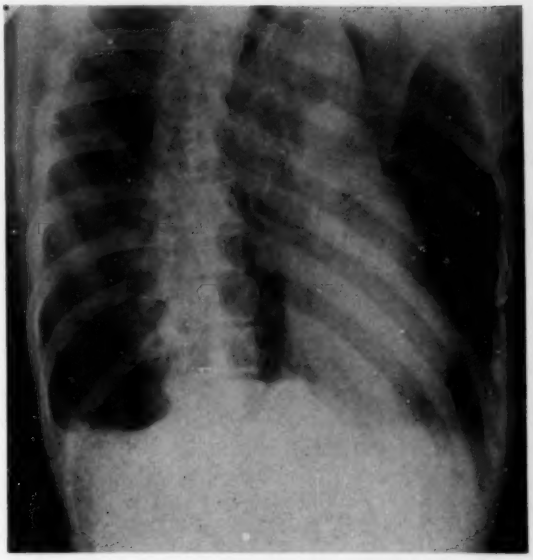

Fig. 5. Case 11. Right oblique view, showing the close relation of the tumor to the diaphragm.

Following the operation there was no appreciable change in the patient's condition. He was discharged from the hospital and did not return for further treatment.

Case 11. E. M., white male, aged forty-eight, a war veteran, was admitted to the University Hospital in January, 1940, for a tonsillectomy. Because the patient complained also of recurring pain in the right lower chest, which occa-

sionally was quite severe, and increased on respiration, a roentgenogram of the chest was made. This showed a round, sharply defined shadow at the base of the right lung field, moving with the diaphragm on deep inspiration. On oblique views this shadow localized slightly posteriorly from the dome of the diaphragm, from which it could not be separated.

A diagnostic pneumothorax and pneumoperitoneum were advised to determine definitely the relation of this tumor to the diaphragm. The patient refused to submit to this examination and we were unable to confirm our diagnosis.

We include this case in the present report because the clinical history and the roentgenograms are similar to those found in other cases of diaphragmatic tumors found in the literature.

CONCLUSIONS

1. Two cases of diaphragmatic tumor have been presented.

2. The clinical symptomatology has been briefly summarized.

3. The differential diagnosis has been discussed.

4. The roentgen diagnostic criteria and the significance of pneumothorax and pneumoperitoneum as integral constituents of the roentgen examination have been stressed.

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ROENTGEN PELVIMETRY IN LABOR BY THE PELVIC INLET GRID METHOD*

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THIS study was conducted in association with the obstetric delivery of 1,420 women, 510 white and 910 negro. About two-thirds were home deliveries, but pathologic conditions were taken care of in the hospital where 138 had pelvic measurements made during labor. This was a single view of the inlet showing the outline of fetal head generally framed within that of the pelvic inlet, since the patient is in a semi-sitting position when the film is exposed, and the fetal head has a tendency to sink into the true pelvis.

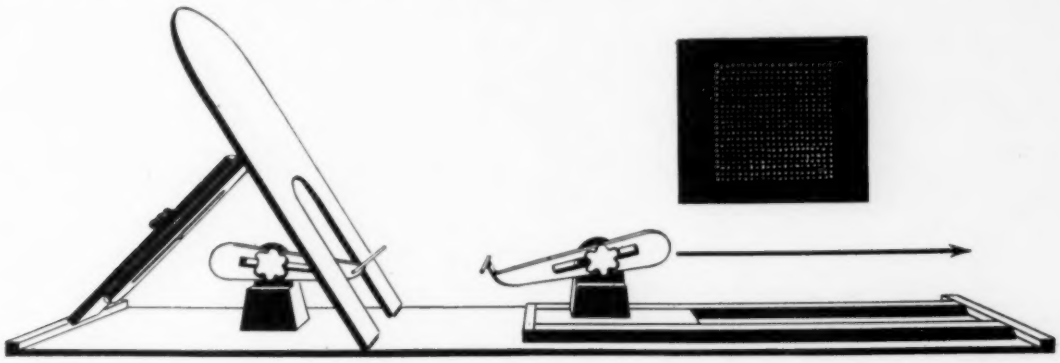

FIG. 1

The resulting 8 by 10 inch film, made with the device previously described,[1] illustrated in Figure 1, shows (1) the outline of the pelvic inlet and its diameter in centimeters directly by counting the dots between two points; (2) the contour of the inlet, and this is important in the pelvic classification into the four groups of Caldwell, Moloy and D'Esopo,[2] who amplified the older classification of the anthropologists, Weber, Stein and others. These types are (*a*) gynecoid (female type) symmetrical, oval pelvic inlet with transverse diameter up to 3 cm. longer than the anteroposterior diameter; (*b*) anthropoid—round or oval inlet with transverse diameter less or no more than equal to the anteroposterior diameter; (*c*) android (male type) inlet with triangular contour wider in the posterior third and narrow anteriorly. In some cases the ischial spines protrude inward and show in the film; (*d*) platypelloid (flat pelvis) in which the anteroposterior diameter is shorter by 3 cm. or more than the transverse diameter. Other characteristics of the four types may be noted by palpating the contour of the sacrum which may bend rather forward, especially in the android type. By palpating the ischial spines and the tuberosities, narrowness of the outlet may be identified, frequently with anthropoid or android pelves. However, from this study these rarer types of pelves seem to have less effect upon labor than does the shortening of the true conjugate (the anteroposterior diameter of the inlet). Of course the effect is greater if there is an associated narrow transverse diameter.

TECHNIQUE

The number of patients in each group in this series is shown in Table 1.

Among the 1,420 deliveries occurred 24

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forceps operations in which the pelvis had roentgen studies, and this included almost all forceps cases. This reveals a forceps incidence of not over 3 per cent and corresponds well with the more or less ideal rate in vogue at the Grady Hospital in Atlanta. Since 16 of the forceps deliveries were in negro. There were 3 low forceps deliveries in white women and 5 in negro women. In 5 cases of forceps delivery there was a narrow outlet as shown by the shortening of distance between the ischial spines seen in the pelvic inlet film and by rectal palpation. Ten of the 24 women who had to be deli-

TABLE I

Gynecoid								Totals
Conjugate vera in cm. White	8–9	9–10 3	10–11 12	11–12 20	12–13 10	13–14 1	14–15	46
Negro	1	10	24	16	5			56
Anthropoid								
Conjugate vera in cm. White	8–9	9–10	10–11	11–12	12–13	13–14	14–15	
Negro			3	6	5			14
Android								
Conjugate vera in cm. White	8–9	9–10 1	10–11 1	11–12 4	12–13 1	13–14	14–15 1	8
Negro		4	1	3				8
Platypelloid								
Conjugate vera in cm. White	8–9 1	9–10	10–11 2	11–12	12–13	13–14	14–15	3
Negro	2							2
Nägele								
Conjugate vera in cm. White	8–9	9–10	10–11	11–12	12–13	13–14	14–15	
Negro		1						1
							(White 57 plus Negro 81)	138

negro women, the incidence is the same as in white women, identical rules as to necessity of application being used in both cases. Table 1 shows a higher incidence of contracted pelves in negro women. It seems, however, that the negro newborn's head is softer and molds more than that of the white infant. The mid forceps incidence was 16 to low forceps 8. Five of the low forceps deliveries were in white women and 11 in vered by forceps had normal size pelves, 2 of these being justo major 12 by 13 cm.; one of these had a 9 pound, 4 ounce baby and the other, 7 pounds 12 ounces.

Among the 24 operative deliveries requiring forceps, 17 of the pelves were gynecoid in type, 3 were anthropoid—all negro, 3 android or tendency to android—2 white and 1 negro, and 1 Nägele pelvis in a negro woman.

Experience teaches one to suspect difficulty in labor in the short obese type of patient, so-called dystrophia dystocia syndrome, and one gathers from the literature that these patients have a tendency to contracted pelves of android or anthropoid types. In this group there were 8 such patients delivered in the hospital, 4 white and 4 negro. All but one had gynecoid type pelves; contraction below 10 cm. anteroposterior in 2, slight contraction 10–11 cm. anteroposterior in 3, large gynecoid pelvis in one and a contracted android type in one negro.

Of these 8 patients, 4 delivered spontaneously—1 negro, contracted gynecoid; 1 negro, slightly contracted gynecoid in 113 hours; 1 white woman of 235 pounds weight with justo major gynecoid pelvis in 3½ hours, and 1 negro whose slightly contracted pelvis had a tendency to android type and a narrow outlet. There were 2 mid forceps, 1 white gynecoid slightly contracted and 1 white normal size gynecoid type. There was 1 cesarean section in a white woman with a slightly contracted gynecoid pelvis who had a thirty hour labor test. There was 1 craniotomy in a negro with a contracted gynecoid pelvis who had a large baby weighing more than 8 pounds.

In this series it is felt that no woman or infant was neglected in giving each mother, under amnesia, full opportunity to complete the labor spontaneously. The craniotomy cases were neglected before admission.

INDIVIDUAL DESCRIPTION OF LABOR IN EACH CASE ILLUSTRATED

The case histories correspond in number to the pelvic outlines shown in Figures 2, 3, 4 and 5.

Case 1. White primipara, aged eighteen; contracted gynecoid pelvis; anteroposterior 9.25 cm., transverse 11 cm. Labor 8 hr. test followed by low cervical cesarean section. Weight of infant 5 lb. 11½ oz.

Case 2. Negro primipara, aged fifteen; contracted gynecoid pelvis; anteroposterior 9.25 cm., transverse 10.25 cm. Labor 1st stage 17 hr., 45 min., 2nd stage 30 min. Presentation left occipito-anterior, weight of infant 8 lb. 5 oz. The measurements of the biparietal and suboccipital bregmatic diameters of the head of the newborn infant were about 0.25 cm. less than those of the pelvic inlet. One year later she duplicated the procedure delivering spontaneously an 8 lb. 4 oz. infant after a rather longer 1st stage and a 30 min. 2nd stage of labor. This proves that Nature is able to accommodate the fetal head to a much tighter fitting pelvis than is usual.

Case 3. Negro, para 0, gravida 1, aged nineteen, toxemia of pregnancy; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11 cm. Labor 1st stage 22 hr., 2nd stage 2 hr. Spontaneous deliveryleft occipito-anterior, living infant, weight 7 lb.

Case 4. Negro primipara, aged seventeen, obese, eclamptic; contracted gynecoid pelvis; anteroposterior 9 cm., transverse 11.5 cm. Labor short after two convulsions. Spontaneous delivery of living infant right occipito-anterior, weight of infant, 7 lb. ½ oz.

Case 5. Negro primipara, aged twenty; gynecoid pelvis; anteroposterior 11 cm., transverse 11.5 cm. Labor 1st stage 33 hr., 2nd stage 4 hr. 35 min. Spontaneous delivery left occipito-anterior, living infant, 7 lb.

Case 6. Negro, para 7, gravida 8, aged thirty-eight, 5 living children, 1 forceps delivery; anthropoid pelvis, anteroposterior 11.5 cm., transverse 11 cm. Test of labor 24 hr. then low cervical cesarean operation with hysterectomy, presentation left occipitoposterior at operation.

Case 7. Negro primipara, aged eighteen; gynecoid pelvis; anteroposterior 12 cm., transverse 12.5 cm. Labor 1st stage 48 hr. with infrequent uterine contractions, 2nd stage 2 hr. Spontaneous delivery, left occipito-anterior, living infant, weight 6 lb. 3 oz.

Case 8. Negro, para 0, gravida 4; markedly contracted gynecoid pelvis with prominent promontory; anteroposterior 8.5 cm., transverse 11.5 cm. Labor 22 hr. test without engagement of the head then low cervical cesarean section, delivery of living infant, weight 8 lb. 5 oz. (congenital syphilis).

Case 9. White primipara, aged twenty-three; gynecoid pelvis; anteroposterior 11.5 cm., transverse 13 cm. Labor 1st stage 25 hr., 2nd stage 40 min. Spontaneous delivery left occipito-anterior, living infant, weight 6 lb. 8¾ oz.

Case 10. Negro primipara, aged nineteen; gynecoid contracted pelvis; anteroposterior 9.5 cm., transverse 11.5 cm. Labor 1st stage 3 or 4 days with infrequent pains, 2nd stage 3½ hr. Low Kjelland forceps delivery, right occipito-anterior, living infant, weight 5 lb. 14 oz.

Case 11. Negro, para 2, gravida 3, aged thirty; gynecoid pelvis; anteroposterior 11.5 cm., transverse 12.25 cm. Labor 1st stage 5 hr. 45 min., 2nd stage 1 hr. Spontaneous delivery, left occipito-anterior, living infant, weight 7 lb. 7 oz.

Case 12. Negro, para 3, gravida 4, aged twenty; gynecoid pelvis; anteroposterior 11.5 cm., transverse 12 cm. Transverse presentation at time of roentgen examination. Fetus turned spontaneously and delivered normally after rapid labor, left occipito-anterior, weight of infant 8 lb.

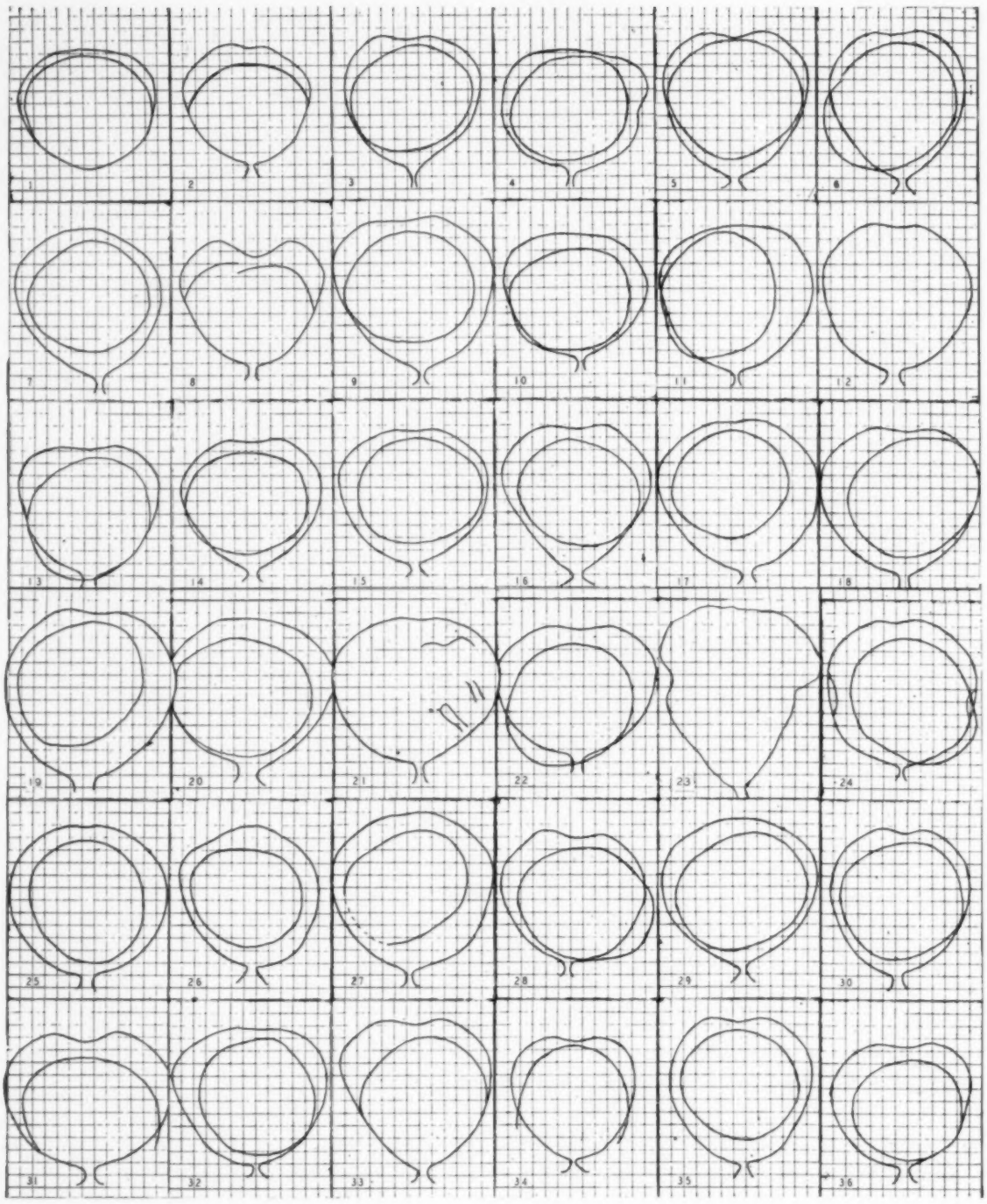

Fig. 2. Pelvic outlines of Cases 1 to 36.

Case 13. White, para 1, gravida 2, aged twenty-one; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11.5 cm. Labor placenta previa marginalis. Voorhees bag inserted into lower uterine segment followed in 1½ hr. by spontaneous delivery living infant, right occipito-anterior, weight 6 lb. 11½ oz.

Case 14. Negro primipara, aged fourteen; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11.25 cm. Labor 1st stage 15 hr. 20 min.,

2nd stage 2 hr. 50 min. Spontaneous delivery, right occipito-anterior, living infant, weight 6 lb. 11 oz.

Case 15. Negro primipara, aged fourteen, anemic with placenta previa marginalis; slight contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 12 cm. Labor 77 hr. Right occipitoposterior rotation 135° to left occipito-anterior with spontaneous delivery living infant, weight 5 lb. 13 oz.

Case 16. White primipara, aged sixteen; android pelvis; anteroposterior 11 cm., transverse 12 cm. Labor 1st stage 48 hr., 2nd stage 7 hr. 31 min. Right occipitoposterior to right occipito-anterior spontaneous delivery living infant, weight 6 lb. 4 oz.

Case 17. Negro primipara, aged twenty-one; gynecoid pelvis; anteroposterior 11.75 cm., transverse 13 cm. Labor 1st stage 6 hr. 30 min., 2nd stage 3 min. Right occipito-anterior spontaneous delivery living infant, weight 6 lb.

Case 18. Negro, para 6, gravida 7, aged thirty; gynecoid pelvis; anteroposterior 11.25 cm., transverse 13 cm. Labor 1st stage 13 hr. 33 min., 2nd stage 2 min. Spontaneous delivery left occipito-anterior, living infant, weight 8 lb. 3 oz.

Case 19. White, para 3, gravida 4, aged twenty-two; justo major gynecoid pelvis. Labor 1st stage 31 hr. 40 min., 2nd stage 2 min. Fetal head ballotable until just before delivery. Spontaneous delivery from complete occiput posterior presentation, living infant, weight 7 lb. 11.5 oz.

Case 20. White, para 2, gravida 3, aged twenty-one; gynecoid pelvis; anteroposterior 11.5 cm., transverse 13.5 cm. Labor 1st stage 22 hr., 2nd stage few minutes. Spontaneous delivery right occipito-anterior, living infant, weight 8 lb. 4 oz.

Case 21. White primipara, aged thirty-three; gynecoid pelvis; anteroposterior 11.5 cm., transverse 13.5 cm. Labor twins, female, small, both breech spontaneous delivery living infants, weight No. 1, 4 lb. 11 oz., No. 2, 4 lb. 8¼ oz.

Case 22. White primipara, aged nineteen; slightly contracted platypelloid pelvis; anteroposterior 10 cm., transverse 13 cm. Labor 1st stage 15½ hr., 2nd stage 2 hr. Spontaneous delivery, right occipito-anterior, living infant, weight 6 lb. 13½ oz.

Case 23. White para 7, gravida 10, aged thirty-six; large android pelvis; anteroposterior 14 cm., transverse 13 cm. Labor, total 3 hr.; precipitate delivery from direct occiput posterior presentation, one living infant, weight 9 lb. 4 oz. One year previously she had spontaneous delivery, from left occipito-anterior of one living infant, weight 9 lb. 13 oz. Total labor, 4 hr. 22 min.

Case 24. White, para 1, gravida 2, aged twenty-five; gynecoid pelvis; anteroposterior 11.5 cm., transverse 12 cm. Labor 1st stage 13 hr., 2nd stage 2 hr. 47 min. Spontaneous delivery, left occipito-anterior, living infant, weight 7 lb. 11¼ oz.

Case 25. Negro, para 2, gravida 3, aged twenty; gynecoid pelvis; anteroposterior 11.75 cm., transverse 12.75 cm. Labor 1st stage 19 hr. 40 min., 2nd stage 28 min. Spontaneous delivery.

Case 26. Negro primipara, aged fifteen; gynecoid pelvis; anteroposterior 11 cm., transverse 11.25 cm. Labor short, entered the hospital with the fetal head in the perineum and spontaneously delivered a living infant from left occipito-anterior, weight 5 lb. 11 oz.

Case 27. White primipara, aged nineteen; justo major gynecoid pelvis; anteroposterior 12.5 cm., transverse 13 cm. Labor 1st stage 31 hr. 14 min., 2nd stage 1 hr. Spontaneous delivery right occipito-anterior living infant, 5 lb. 3 oz.

Case 28. White primipara, aged thirty-five; slight contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11.5 cm. Labor 1st stage 1 hr., 2nd stage 4 hr. 32 min. (left hand prolapsed beside head). Spontaneous delivery, right occipito-anterior living infant, weight 7 lb. 4¼ oz.

Case 29. Negro primipara, aged seventeen, mild toxemia; gynecoid pelvis; anteroposterior 11.5 cm., transverse 12.5 cm. Labor 1st stage 27 hr. 15 min., 2nd stage 3 hr. 9 min. Low forceps right occipito-anterior living infant, weight 7 lb. 9 oz.

Case 30. Negro primipara, aged twenty-four, toxemia; anthropoid pelvis; anteroposterior 11 cm., transverse 11 cm. Labor 1st stage 36 hr. Cervix fully dilated by a Voorhees bag. 2nd stage 1 hr. 30 min. Mid pelvis Kjelland application of Kjelland forceps. O.L.T. living infant, weight 7 lb. 14 oz.

Case 31. White, para 5, gravida 6, aged twenty-five; slight contracted platypelloid pelvis; anteroposterior 10 cm., transverse 13.5 cm. Labor 1st stage 14 hr. 45 min., 2nd stage 10 min. Spontaneous delivery, right occipito-anterior living infant, weight 8 lb. 4 oz.

Case 32. White, para 5, gravida 6, aged thirty-two; gynecoid pelvis; anteroposterior 10.75 cm., transverse 12.5 cm. Labor 1st stage 7 hr. 30 min., 2nd stage 30 min. Spontaneous delivery, right occipitoposterior to right occipito-anterior living infant, 7 lb. 4 oz.

Case 33. Negro, para 5, gravida 6, aged thirty-four; android pelvis; anteroposterior 11.25 cm., transverse 12 cm. Labor 1st stage 10 hr., 2nd stage 25 min. Spontaneous delivery O.R.A. living infant, weight 8 lb.

Case 34. Negro primipara, aged fifteen; contracted pelvis android tendency; anteroposterior 9 cm., transverse 10 cm. Labor 1st stage 22 hr., 2nd stage 2 hr. Spontaneous delivery O.R.A. living infant, weight 6 lb. 8 oz.

Case 35. Negro primipara, aged eighteen, toxemia; gynecoid pelvis; anteroposterior 11 cm., transverse 12.25 cm. Labor 1st stage 15 hr. 30 min., 2nd stage 1 hr. 25 min. Spontaneous delivery O.R.A. living infant, weight 6 lb. 12 oz.

Case 36. Negro primipara, aged eighteen; contracted gynecoid pelvis; anteroposterior 9.25 cm., transverse 11 cm. Labor 1st stage 19 hr. 15 min., 2nd stage 30 min. Spontaneous delivery O.R.A. living infant, weight 7 lb. 4½ oz.

Case 37. Negro primipara, aged thirty-two; gynecoid pelvis; anteroposterior 11 cm., transverse 12.75 cm. Labor, precipitant spontaneous delivery of living infant.

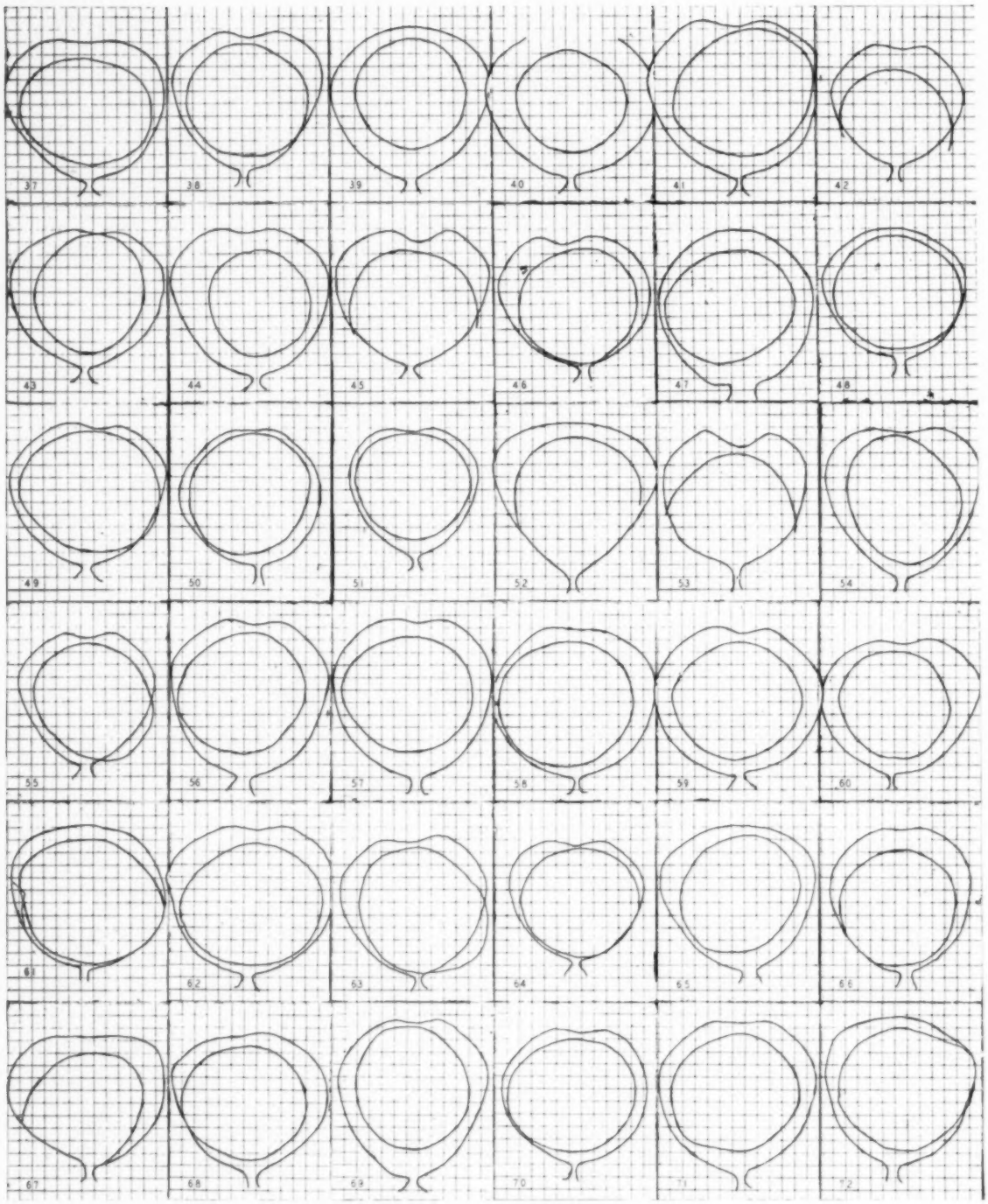

Fig. 3. Pelvic outlines of Cases 37 to 72.

Case 38. White, para 2, gravida 3, aged thirty-seven; slight contracted gynecoid pelvis; anteroposterior 10.25 cm., transverse 12 cm. Labor 1st stage 7 hr., 2nd stage 1 hr. Spontaneous delivery O.R.A. living infant, weight 7 lb. 2 oz.

Case 39. White, para 1, gravida 2, aged nineteen, mild toxemia; gynecoid pelvis; anteroposterior 12 cm., transverse 13 cm. Labor 1st stage 6 hr. 30 min., 2nd stage 10 min. Spontaneous delivery O.R.A. living infant, weight 6 lb. 7½ oz.

Case 40. White primipara, aged eighteen; gynecoid pelvis; anteroposterior 11 cm., transverse 13.5 cm. Labor 1st stage 8 hr. 50 min., 2nd stage 30 min. Spontaneous delivery O.R.A. living infant, weight 5 lb. 12 oz.

Case 41. White, para 8, gravida 9, aged forty-three, obese and toxemic; justo major gynecoid pelvis; anteroposterior 12 cm., transverse 13.5 cm. Labor 1st stage futile uterine contractions over period of 3 days during which time she received 25 gm. $MgSO_4$ for hypertension. Finally cervix became fully dilated and she spontaneously delivered O.L.A. living infant, weight 9 lb. 8 oz.

Case 42. Negro primipara, aged twenty-five, toxemia; contracted gynecoid pelvis; anteroposterior 9.25 cm., transverse 10.75 cm. Labor 1st stage 76 hr., 2nd stage 3 hr. Deep transverse arrest in mid pelvis O.R.T. Kjelland application of Kjelland forceps delivered living infant, weight 6 lb. 12 oz.

Case 43. Negro, para 2, gravida 3, aged twenty-three; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 12.25 cm. Labor, 1st stage 39 hr., 2nd stage 4 hr. 35 min. Low forceps delivery O.L.A. living infant, weight 6 lb. 7 oz.

Case 44. White primipara, aged sixteen; gynecoid pelvis; anteroposterior 12.0 cm., transverse 12.5 cm. Labor, 1st stage 3 hr., 2nd stage 50 min. Spontaneous delivery O.R.A. living infant, weight 6 lb. 1 oz.

Case 45. Negro primipara, aged seventeen; slightly contracted gynecoid pelvis with prominent promontory; anteroposterior 10 cm., transverse 12.5 cm. Labor, 1st stage 24 hr., 2nd stage 1 hr. Spontaneous delivery O.R.A. living infant.

Case 46. White, para 1, gravida 2, aged twenty-two; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 12 cm. Labor, 1st stage 26 hr. 45 min., 2nd stage 1 min. Spontaneous delivery O.R.P. living infant, weight 8 lb. 4½ oz.

Case 47. Negro, para 6, gravida 7, aged thirty-four, toxemia; justo major gynecoid pelvis; anteroposterior 12 cm., transverse 12.5 cm. Labor, 1st stage 5 hr., 2nd stage 10 min. Spontaneous delivery O.L.A. living infant, weight 9 lb. 12 oz.

Case 48. Negro, para 2, gravida 3, aged twenty, toxemia; slight contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11.5 cm. Labor, 1st stage 19 hr., 2nd stage 10 min. Spontaneous delivery O.R.A. living infant, weight 8 lb. 2 oz.

Case 49. White, para 1, gravida 2, aged eighteen, toxemia; gynecoid pelvis; anteroposterior 10.75 cm., transverse 12.5 cm. Labor, 1st stage 11 hr. 15 min., 2nd stage 27 min. Spontaneous delivery O.L.A. living infant, weight 7 lb. 12¼ oz. (1st labor 48 hr. delivered by Kjelland forceps mid pelvis O.L.T. living infant, weight 7 lb. 8 oz.)

Case 50. Negro, para 4, gravida 5, aged twenty-four; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 11.25 cm. Labor, 1st stage 8 hr., 2nd stage 20 min. Spontaneous delivery O.L.A. living infant, weight 5 lb. 13 oz.

Case 51. Negro, para 4, gravida 5, aged thirty-four, toxemia; contracted gynecoid pelvis; anteroposterior 9.75 cm., transverse 10.25 cm. Labor, 1st stage test 33 hr. 50 min. then low cervical cesarean section. Fetal head tightly fitted into pelvic brim O.R.T. living infant, weight 8 lb. 8 oz. (All of her previous children were living.)

Case 52. White, para 2, gravida 3, aged twenty-seven; large android pelvis; anteroposterior 11.5 cm., transverse 13 cm. Labor, 1st stage 22 hr. 30 min., 2nd stage 2 hr. 25 min. Spontaneous delivery O.L.A. living infant, weight 8 lb. 8⅝ oz.

Case 53. White, para 2, gravida 3, aged twenty-nine; slightly contracted gynecoid pelvis with prominent promontory; anteroposterior 9.5 cm., transverse 11.5 cm. Labor, 1st stage 9 hr. 15 min., 2nd stage 31 min. Spontaneous delivery O.L.A. living infant, weight 9 lb. 8 oz.

Case 54. Negro, para 1, gravida 2, aged twenty-six; android pelvis; anteroposterior 11.5 cm., transverse 12.25 cm. Labor, 1st stage 15 hr. 30 min., 2nd stage 2 hr. 35 min. Spontaneous delivery O.L.A. living infant, weight 9 lb. 8 oz.

Case 55. Negro primipara, aged seventeen, dystrophia dystocia syndrome, obese and toxemic; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11 cm. Labor, 1st stage 112 hr., 2nd stage 1 hr. Spontaneous delivery O.R.P. to O.R.A. living infant, weight 6 lb.

Case 56. White, para 2, gravida 3, aged twenty-one; justo major gynecoid pelvis; anteroposterior 12 cm., transverse 12.75 cm. Labor, 1st stage 4 hr. 45 min., 2nd stage 15 min. Spontaneous delivery O.L.A. living infant, weight 6 lb. 12 oz.

Case 57. Negro, para 2, gravida 3, aged twenty; justo major gynecoid pelvis; anteroposterior 12.25 cm., transverse 12.75 cm. Labor, 1st stage 11 hr. 40 min., 2nd stage 10 min. Spontaneous delivery O.L.A. living infant, weight 6 lb. 8 oz.

Case 58. White primipara, aged seventeen; gynecoid pelvis; anteroposterior 11.5 cm., transverse 13 cm. Labor, 1st stage 19 hr. 30 min., 2nd stage 2 hr. 10 min. Spontaneous delivery O.L.A. living infant, weight 9 lb. 14 oz.

Case 59. White primipara, aged fifteen; gynecoid pelvis; anteroposterior 11.5 cm., transverse 13.5 cm. Labor, 1st stage 10 hr., 2nd stage 1 hr. 10 min. Spontaneous delivery O.L.A. living infant, weight 7 lb. 4½ oz.

Case 60. White primipara, aged twenty-eight; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 13 cm. Labor, 1st stage 25 hr. 55 min., 2nd stage 39 min. Spontaneous delivery O.L.A. living infant, weight 4 lb. 4¼ oz.

Case 61. White, para 1, gravida 2, aged twenty-nine; gynecoid pelvis; anteroposterior 11 cm., transverse 12 cm. Labor, 1st stage 12 hr. 5 min., 2nd stage 1 hr. 40 min. Spontaneous delivery O.L.A. living infant, weight 10 lb. 6⅞ oz.

Case 62. White, para 2, gravida 3, aged nineteen, toxemia and twins; gynecoid pelvis; anteroposterior 11.5 cm., transverse 13 cm. Labor, 1st stage 17 hr.

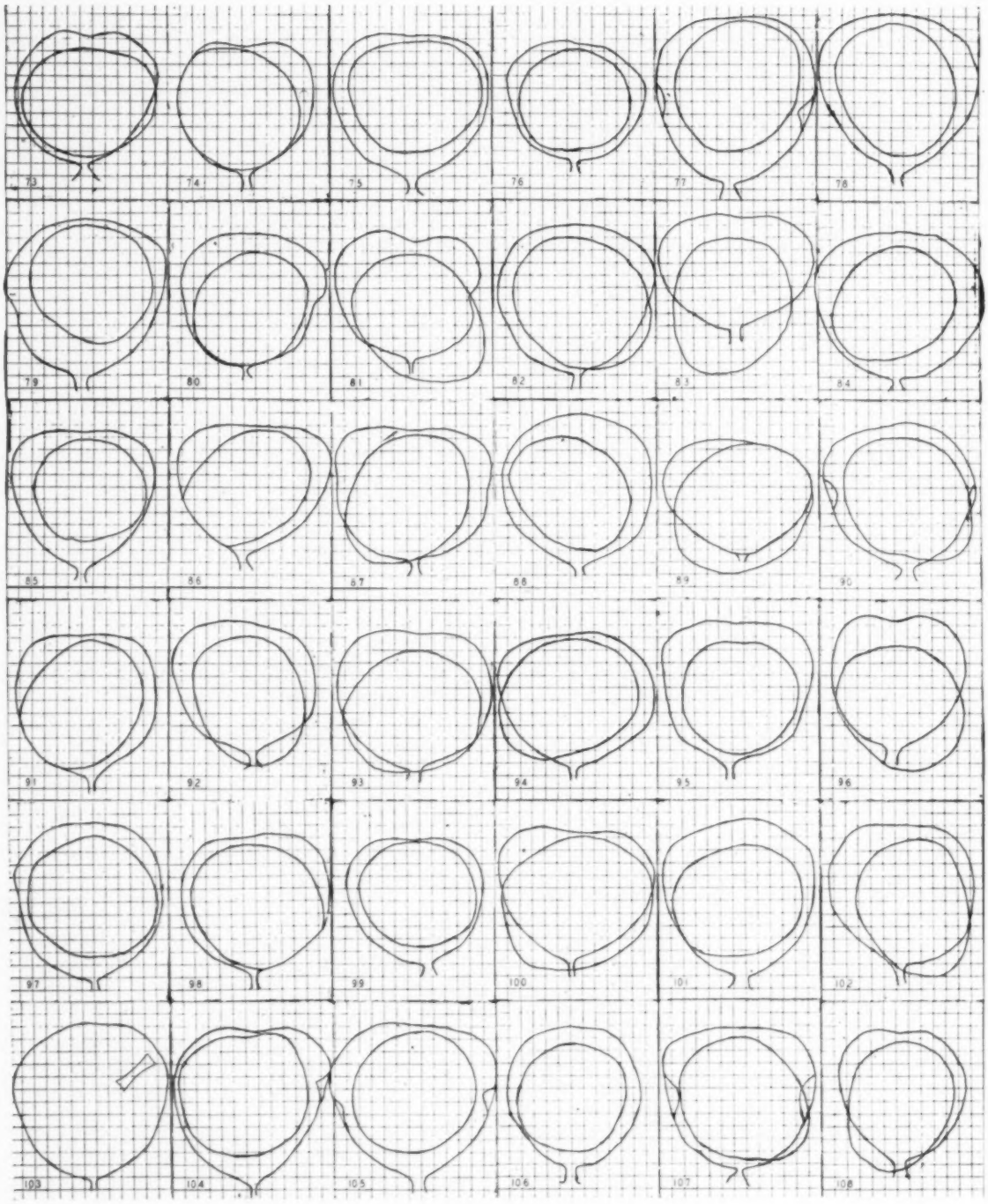

Fig. 4. Pelvic outlines of Cases 73 to 108.

5 min., 2nd stage 1 hr. Spontaneous delivery O.L.A. living female, weight 6 lb. 3 oz., and O.L.A. living male, weight 6 lb. 2 oz.

Case 63. White, para 2, gravida 3, aged twenty-one; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 11.5 cm. Labor, 1st stage 17 hr. 50 min., 2nd stage 2 hr. 10 min. Spontaneous delivery O.R.A. living infant, weight 8 lb. 4 oz.

Case 64. Negro primipara, aged seventeen, dystrophia dystocia syndrome; contracted gynecoid pelvis; anteroposterior 9 cm., transverse 10.5 cm. Labor,

1st stage 63 hr., 2nd stage 2 hr. 5 min. Maternal temperature 104° F., pulse 180 in spite of fluids and oxygen. Dead fetus delivered by craniotomy. Weight of fetus 6 lb. 11 oz plus an estimated 20 per cent for brains and blood.

Case 65. Negro, para 1, gravida 2, aged nineteen; pelvis android tendency; anteroposterior 11.5 cm., transverse 12.5 cm. Labor, 1st and 2nd stages 18 hr. Spontaneous delivery O.L.P. to O.L.A. living infant, weight 6 lb. 14 oz.

Case 66. Negro primipara, aged nineteen; anthropoid pelvis; anteroposterior 11.5 cm., transverse 11.5 cm. Labor, 1st stage 15 hr., 2nd stage 4 hr. 27 min. Kjelland mid forceps delivery O.L.T. living infant, weight 7 lb.

Case 67. White primipara, aged thirty-four, short and stout; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 12.5 cm. Labor, 1st stage 38 hr. 30 min., 2nd stage 8 hr. 52 min. Mid forceps delivery O.L.P. living infant, weight 7 lb. 5 oz.

Case 68. Negro primipara, aged sixteen; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 12.5 cm. Labor, 1st stage 19 hr., 2nd stage 8 hr. 18 min. Delay due to large shoulders. Low forceps delivery O.L.A. living infant, weight 8 lb. 6 oz.

Case 69. Negro primipara, aged seventeen; anthropoid pelvis; anteroposterior 12.25 cm., transverse 12 cm. Labor, 1st and 2nd stages 6 hr. Spontaneous delivery O.R.A. living infant, weight 6 lb. 8 oz.

Case 70. Negro primipara, aged twenty-one; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 11.5 cm. Labor, 1st stage 46 hr., 2nd stage 2 hr. 50 min. Spontaneous delivery O.L.T. to O.L.A. living infant, weight 7 lb. 12 oz.

Case 71. White primipara, aged eighteen; gynecoid pelvis; anteroposterior 11.5 cm., transverse 12.75 cm. Labor, 1st stage 15 hr. 12 min., 2nd stage 1 hr. 16 min. Spontaneous delivery O.L.T. to O.L.A. living infant, weight 6 lb. 5 oz.

Case 72. Negro, para 1, gravida 2, aged twenty-seven, toxemia; gynecoid pelvis; anteroposterior 11.75 cm., transverse 12 cm. Labor, 1st stage 11 hr. 45 min., 2nd stage 8 hr. 35 min. Extension of the fetal head, mid pelvis application of Kjelland forceps O.R.P. to O.R.A. delivery of stillborn infant, weight 10 lb. 3 oz. Autopsy of the fetus revealed brain injury. (History of 1st labor: 1st stage 60 hr., 2nd stage 5 hr. 48 min. Kjelland forceps to fetal head in mid pelvis O.L.T. delivery living infant, weight 8 lb. 3 oz.)

Case 73. Negro, para 3, gravida 4, aged thirty-six; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11.25 cm. Labor, 1st stage 24 hr., 2nd stage 15 min. Spontaneous delivery O.L.A. living infant, weight 6 lb. 12 oz.

Case 74. Negro, para 4, gravida 5, aged twenty-three, toxemia; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 10.75 cm. Labor, 1st stage 2 hr. 40 min., 2nd stage 20 min. Spontaneous delivery O.L.A. living infant, weight 9 lb. 11 oz.

Case 75. Negro, para 4, gravida 5, aged twenty-seven; gynecoid pelvis; anteroposterior 11 cm., transverse 12.5 cm. Labor, 1st stage 32 hr., 2nd stage short. Spontaneous delivery living infant O.L.A., 7 lb. 3 oz.

Case 76. Negro primipara, aged sixteen; contracted gynecoid pelvis; anteroposterior 9 cm., transverse 11 cm. Labor, 1st stage 33 hr., 2nd stage 1 hr. 50 min. Spontaneous delivery living infant, weight 5 lb. 12 oz.

Case 77. White, para 0, gravida 2, aged eighteen; justo major gynecoid pelvis; anteroposterior 12.75 cm., transverse 13 cm. Labor, 1st stage 17 hr., 2nd stage 20 min. Spontaneous delivery O.L.A. living infant, weight 8 lb.

Case 78. White primipara, aged twenty-one; gynecoid pelvis with android tendency; anteroposterior 12 cm., transverse 12.75 cm. Labor, 1st stage 7 hr., 2nd stage 31 min. Spontaneous delivery O.L.A. living infant, weight 8 lb. 10 oz.

Case 79. White primipara, aged eighteen; gynecoid pelvis; anteroposterior 12 cm., transverse 13 cm. Labor, 1st stage 36 hr., 2nd stage 5 hr. 27 min. Mid pelvis Kjelland forceps O.L.T. living infant, weight 7 lb. 12 oz.

Case 80. Negro primipara, aged eighteen; slightly contracted gynecoid pelvis; anteroposterior 10.25 cm., transverse 11.75 cm. Labor, 1st stage 34 hr. 30 min., 2nd stage 30 min. Spontaneous delivery O.L.A. living infant, weight 5 lb. 2 oz.

Case 81. White primipara, aged fifteen; contracted gynecoid pelvis; anteroposterior 9 cm., transverse 11.5 cm. Labor, 1st stage 52 hr. 28 min., 2nd stage 1 hr. 56 min. Low forceps delivery O.R.A. living infant, weight 7 lb. 6 oz.

Case 82. White, para 1, gravida 2, aged twenty-one; gynecoid pelvis; anteroposterior 11.5 cm., transverse 12.5 cm. Labor, 1st stage 6 hr. 20 min., 2nd stage 27 min. Spontaneous delivery O.R.A. living infant, weight 8 lb. 13 oz.

Case 83. White, para 2, gravida 3, aged twenty-one; contracted platypelloid pelvis; anteroposterior 8.75 cm., transverse 12.75 cm. Labor, 20 hr. without engagement, then low cervical cesarean section at which there was found a brow presentation occiput to the right. Living infant, weight 7 lb. 3¼ oz. (She had had two previous difficult spontaneous deliveries, both babies were small.)

Case 84. Negro primipara, aged sixteen; gynecoid pelvis; anteroposterior 11.5 cm., transverse 13.5 cm. Labor, 1st stage 42 hr., 2nd stage 2 hr. 40 min. Spontaneous delivery O.L.A. living infant, weight 5 lb. 2 oz.

Case 85. Negro, primipara, aged nineteen; slightly contracted pelvis android tendency; anteroposterior 10.5 cm., transverse 11.5 cm. Labor, 1st stage 40 hr. 20 min., 2nd stage 28 min. Spontaneous delivery O.R.A. living infant, weight 5 lb. 2 oz.

Case 86. Negro primipara, aged seventeen; slightly contracted gynecoid pelvis, android tendency; anteroposterior 10 cm., transverse 12.25 cm. Labor, 1st stage 22 hr. 45 min., 2nd stage 30 min. Spontaneous delivery O.L.A. living infant, weight 9 lb.

Case 87. White, para 1, gravida 2, aged twenty-three; gynecoid pelvis; anteroposterior 10.5 cm., transverse 12.75 cm. Labor, 1st stage 6 hr. 30 min., 2nd stage 34 min. Spontaneous delivery O.R.A. living infant, weight 6 lb.

Case 88. Negro primipara, aged eighteen; anthropoid pelvis; anteroposterior 12 cm., transverse 12 cm. Labor, 1st stage 36 hr. 30 min., 2nd stage 40 min. Spontaneous delivery O.R.P. to O.R.A. living infant, weight 7 lb. 12 oz.

Case 89. Negro primipara, achondroplastic dwarf, aged twenty-four, brought to hospital after 50 hr. of labor, anemic, eclamptic, neglected, and fetus in poor condition; large fetus and platypelloid pelvis; anteroposterior 8.5 cm., transverse 11.5 cm. Delivery by craniotomy infant, weight 8 lb. 6 oz. plus estimated 20 per cent for brains and blood. Uterus packed, mother died on sixth day, autopsy revealed infarct of the posterior lobe of the pituitary gland, anemia, fatty degeneration of the liver, pulmonary congestion and edema.

Case 90. Negro primipara, aged sixteen; gynecoid pelvis; anteroposterior 11 cm., transverse 12 cm. Labor, 1st stage 36 hr., 2nd stage 1 hr. 30 min. Spontaneous delivery O.R.P. to O.R.A. living infant, weight 6 lb. 6 oz.

Case 91. Negro primipara, aged twenty-five; gynecoid pelvis; anteroposterior 11 cm., transverse 11.5 cm. Labor, 1st stage 30 hr., 2nd stage 2 hr. Spontaneous delivery O.L.P to O.L.A. living infant, weight 7 lb. 5 oz.

Case 92. Negro primipara, aged twenty; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 11.75 cm. Labor, 1st stage 20 hr., 2nd stage 2 hr. 15 min. Spontaneous delivery O.R.A. living infant, weight 6 lb. 4 oz.

Case 93. White, para 2, gravida 3, aged twenty; gynecoid pelvis; anteroposterior 10.5 cm., transverse 12.25 cm. Labor, 1st stage 12 hr. 20 min., 2nd stage 30 min. Spontaneous delivery O.L.A. living infant, weight 6 lb. 14 oz.

Case 94. White primipara, aged sixteen, short fat type, hereditary; slightly contracted gynecoid pelvis; anteroposterior 10.25 cm., transverse 12.75 cm. Labor, 30 hr. test, then low cervical cesarean section. Delivery of living infant, weight 6 lb. 1 oz.

Case 95. Negro, para 6, gravida 7, aged twenty-eight; gynecoid pelvis; anteroposterior 10.75 cm., transverse 12 cm. Labor, 1st stage 23 hr., 2nd stage 25 min. Spontaneous delivery O.R.A. living infant, weight 7 lb. 11 oz.

Case 96. Negro, para 2, gravida 3, aged twenty-seven, toxemia; slight contracted gynecoid pelvis; anteroposterior 10 cm., transverse 10.75 cm. Labor, 1st stage 77 hr., 2nd stage 5 hr. 25 min. Mid forceps O.R.P. stillborn infant, weight 9 lb. 5 oz. Autopsy: brain injury.

Case 97. Negro, para 1, gravida 2, aged twenty-nine; anthropoid pelvis; anteroposterior 12 cm., transverse 12 cm. Labor, 1st stage 48 hr., 2nd stage 30 min. Spontaneous delivery O.L.P. to O.L.A. living infant, weight 8 lb. 12 oz.

Case 98. Negro primipara, aged twenty; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 12 cm. Labor, 1st stage 79 hr. at home with several vaginal examinations. Membranes ruptured and infection of amniotic fluid, dead fetus delivered by craniotomy, mother died suddenly six hours later. Autopsy: bilateral pulmonary emboli, streptococcus found in vaginal culture.

Case 99. Negro primipara, aged nineteen; slightly contracted gynecoid pelvis; anteroposterior 9.75 cm., transverse 11 cm. Labor, 1st stage 30 hr., 2nd stage 6 hr. Kjelland forceps delivery O.R.T. living infant weight 5 lb. 6 oz.

Case 100. Negro, para 2, gravida 4, aged twenty; slightly contracted gynecoid pelvis; anteroposterior 10 cm., transverse 12.5 cm. Labor, 1st stage 56 hr., 2nd stage 2 hr. 20 min. Spontaneous delivery O.L.A. living infant, weight 9 lb.

Case 101. Negro primipara, aged sixteen; anthropoid pelvis; anteroposterior 12.25 cm., transverse 12 cm. Placenta previa marginalis treated by rupture of the membranes. Labor, 1st stage 12 hr. 20 min., 2nd stage 20 min. Spontaneous delivery O.L.A. living infant, weight 5 lb. 4 oz.

Case 102. White primipara, aged eighteen; gynecoid pelvis; anteroposterior 11 cm., transverse 13.75 cm. Labor, 1st stage 9 hr. 30 min., 2nd stage 1 hr. Spontaneous delivery O.R.A. living infant, 7 lb. 6 oz.

Case 103. Negro, para 3, gravida 4, aged thirty, toxemia; gynecoid pelvis; anteroposterior 12 cm., transverse 12.5 cm. Labor, 1st stage 7 hr., 2nd stage 1 hr. 10 min. Breech delivery, macerated fetus, weight 6 lb. 2 oz.

Case 104. White, para 4, gravida 5, aged twenty-nine; malformed gynecoid pelvis; anteroposterior 11.75 cm., transverse 12.5 cm. Labor 1st stage 36 hr., 2nd stage 1 hr. 10 min. Spontaneous delivery O.L.A. living infant, weight 4 lb. 5½ oz.

Case 105. White primipara, aged twenty; gynecoid pelvis; anteroposterior 11.75 cm., transverse 13.25 cm. Labor, 1st stage 8 hr., 2nd stage 1 hr. 48 min. Spontaneous delivery O.L.A. living infant, weight 6 lb. 10 oz.

Case 106. Negro primipara, aged sixteen, toxemia; slightly contracted anthropoid pelvis; anteroposterior 10.75 cm., transverse 10.5 cm. Labor, 1st stage 12 hr. 10 min., 2nd stage 10 min. Spontaneous delivery O.L.A. living infant, weight 5 lb. 4 oz.

Case 107. White primipara, aged fifteen; gynecoid pelvis with narrow outlet; anteroposterior 11 cm., transverse 12.25 cm. Labor, 1st stage 58 hr. 5 min., 2nd stage 53 min. Low forceps delivery after rotation O.R.P. to O.R.A. living infant, weight 6 lb. 12¼ oz.

Case 108. Negro primipara, aged twenty-one, tox-

emia; contracted anthropoid pelvis; anteroposterior 10 cm., transverse 10 cm. Labor, 1st stage 13 hr., 2nd stage 5 hr. 20 min. Kjelland mid forceps O.L.T., infant died following cerebellar injuries.

Case 109. Negro, para 2, gravida 3, aged twenty; anthropoid pelvis; anteroposterior 11 cm., transverse 11 cm. Labor, 1st stage 23 hr. 30 min., 2nd stage 30 min. Spontaneous delivery O.L.A. living infant, weight 6 lb. 6 oz.

Case 110. Negro primipara, aged seventeen, pyelitis of pregnancy and anemia; justo major gynecoid pelvis; anteroposterior 12.5 cm., transverse 13 cm. Labor, 1st stage irregular contractions for 72 hr., 2nd stage 3 hr. Mid forceps delivery O.L.A. living infant, weight 9 lb. 4 oz.

Case 111. Negro primipara, aged seventeen, toxemia; anthropoid pelvis with narrow outlet; anteroposterior 12 cm., transverse 12 cm. Labor, 1st stage 16 hr., 2nd stage 2 hr. Spontaneous delivery O.L.A. living infant, weight 6 lb. 7 oz.

Case 112. White, para 4, gravida 5, aged thirty-six; contracted android pelvis; anteroposterior 9.5 cm., transverse 12 cm. Labor, 1st stage 16 hr., 2nd stage 1 hr. Spontaneous delivery O.R.A. living infant, 5 lb. 12 oz.

Case 113. Negro primipara, aged fifteen; slightly contracted gynecoid pelvis; anteroposterior 10.75 cm., transverse 11.75 cm. Labor, 1st stage 30 hr., 2nd stage 2 hr. plus. Mid Kjelland forceps delivery O.L.T. living infant, weight 7 lb. 8 oz.

Case 114. Negro, para 4, gravida 5, aged twenty-seven, toxemia; slight contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 11.75 cm. Labor, 1st stage 24 hr., 2nd stage 2 hr. Spontaneous delivery O.R.T. to O.R.A. living infant, weight 5 lb. 8 oz.

Case 115. Negro primipara, aged eighteen, toxemia; gynecoid pelvis with narrow outlet; anteroposterior 11 cm., transverse 11.75 cm. Labor, 1st stage 56 hr., 2nd stage 5 hr. 15 min. Kjelland application of Kjelland forceps O.R.P. in mid pelvis, delivery living infant, weight 6 lb. 9 oz. This fetal head rotated from O.L.P. to O.P. to O.R.P.

Case 116. White primipara, aged twenty-six; pelvis tendency to android; anteroposterior 10.75 cm., transverse 11 cm. Labor, 1st stage 14 hr., 2nd stage 21 min. Spontaneous delivery O.R.T. to O.R.A. living infant, weight 6 lb. 3½ oz.

Case 117. White primipara, aged seventeen, female type; pelvis android tendency, very narrow outlet; anteroposterior 11.25 cm., transverse 11.75 cm. Labor, 1st stage 27 hr., 2nd stage 7 hr. 45 min. Mid pelvis Kjelland application of Kjelland forceps O.L.P. delivery of living infant, weight 6 lb. 3 oz., temporary facial paralysis of fetus.

Case 118. White, para 1, gravida 4, aged seventeen, had previous low cervical section; gynecoid pelvis; anteroposterior 11.25 cm., transverse 13 cm Labor, 1st stage 18 hr. 56 min., 2nd stage 1 hr. Spontaneous delivery O.L.A. living infant, weight 7 lb. 2½ oz.

Case 119. White primipara, aged eighteen; gynecoid pelvis narrow outlet; anteroposterior 12 cm., transverse 12.25 cm. Labor, 1st stage 33 hr. 30 min., 2nd stage 10 min. Spontaneous delivery O.L.A. small macerated fetus, dead for several days.

Case 120. Negro, para 4, gravida 5, aged thirty-three; contracted pelvis android tendency; anteroposterior 9.5 cm., transverse 11 cm. Labor, 1st stage 7 hr., 2nd stage 9 hr. 49 min. Dead fetus transverse with elbow presenting, weight 7 lb. 7 oz. delivered by version and extraction.

Case 121. Negro, para 5, gravida 6, aged thirty-five; gynecoid pelvis; anteroposterior 10.5 cm., transverse 13 cm. Labor, 1st stage 13 hr., 2nd stage 52 min. Spontaneous delivery O.L.A. living infant, weight 7 lb. 14 oz.

Case 122. White, para 3, gravida 4, aged twenty-four, familial obesity; justo major gynecoid pelvis slight android tendency; anteroposterior 12.25 cm., transverse 14.25 cm. Labor induced, 1st stage 3 hr. 30 min., 2nd stage 12 min. Spontaneous delivery O.L.A. living infant, weight 10 lb. 1½ oz.

Case 123. White primipara, aged twenty; slightly contracted gynecoid pelvis; anteroposterior 10.25 cm., transverse 12 cm. Labor, 1st stage 22 hr., 2nd stage 6 hr. Low forceps delivery O.R.A. living infant, weight 9 lb. 6 oz.

Case 124. Negro primipara, aged seventeen, toxemia; slightly contracted anthropoid pelvis; anteroposterior 10.75 cm., transverse 10.75 cm. Labor, 1st stage 26 hr. 30 min., 2nd stage 2 hr. 30 min. Spontaneous delivery O.R.A. living infant, weight 6 lb. 1 oz.

Case 125. Negro primipara, aged eighteen, toxemia; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 12 cm. Labor, 1st stage 10 hr. 30 min., 2nd stage 1 hr. Spontaneous delivery O.R.A. living infant, weight 7 lb. 4 oz.

Case 126. Negro, para 1, gravida 2, aged nineteen; slightly contracted gynecoid pelvis; anteroposterior 10.5 cm., transverse 12 cm. Labor, 32 hr. 10 min., test, head engaged, cervix dilated 4 cm. Low cervical cesarean section, living infant, weight 7 lb. 1 oz. This patient had postpartum eclampsia. (Her 1st labor, 1st stage 32 hr. 10 min., 2nd stage 1 hr. 45 min. Spontaneous delivery living infant, weight 7 lb. 4 oz.) This patient might have delivered the 2nd time with the use of the Voorhees bag to dilate the cervix.

Case 127. Negro primipara, aged twenty-two, short obese type; contracted pelvis android tendency with narrow outlet; anteroposterior 9.75 cm., transverse 10.5 cm. Labor, 1st stage 17 hr. 45 min., 2nd stage 1 hr. 10 min. Spontaneous delivery O.R.A. living infant, weight 7 lb.

Case 128. Negro primipara, aged eighteen; Nägele pelvis; anteroposterior 9.75 cm., transverse 10.5 cm. Labor, 1st stage 32 hr., 2nd stage 5 hr. 55 min. Mid pelvis application Kjelland forceps O.L.A. delivery living infant, weight 5 lb. 9 oz., with fetal head biparietal diameter 9 cm. and suboccipital bregmatic diameter 8 cm.

Case 129. Negro, para 4, gravida 5, aged thirty-six, diabetes and marked toxemia; contracted platy-

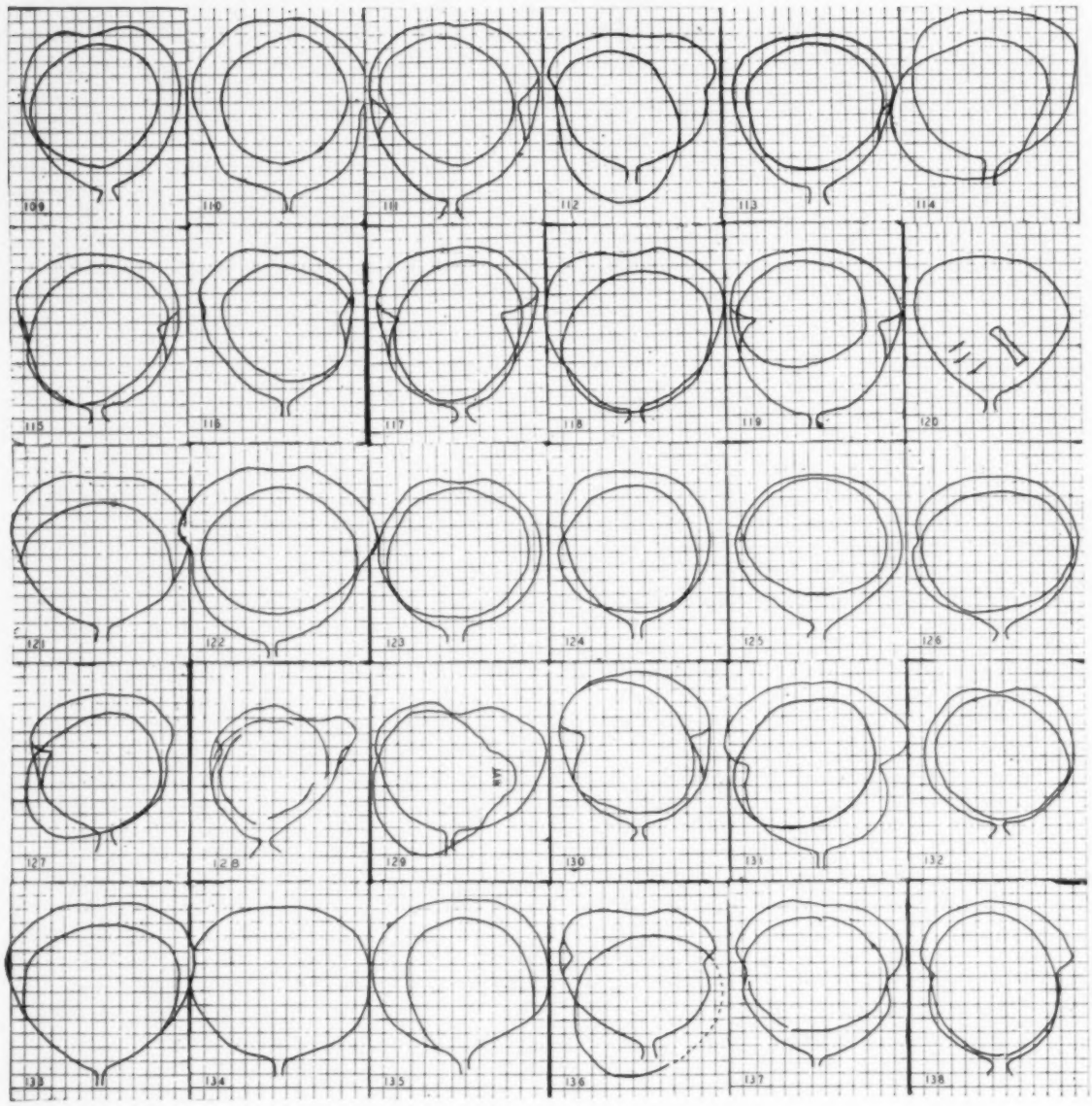

Fig. 5. Pelvic outlines of Cases 109 to 138.

pelloid pelvis; anteroposterior 8.5 cm., transverse 12 cm. She had given birth to 3 babies spontaneously weighing from 6 lb. 1 oz. to 7 lb. Labor, membranes ruptured 22 hr., head engaging, cervix dilated 4 cm. Low cervical cesarean section delivery, living infant, brow presentation, weight 9 lb. 7 oz. The mother died suddenly the next day, autopsy not granted.

Case 130. Negro, para 1, gravida 2, aged thirty, with multiple large fibromyomas; anthropoid pelvis narrow outlet; anteroposterior 11 cm., transverse 11 cm. Labor, 1st stage 14 hr., 2nd stage 50 min. Spontaneous delivery complete occiput posterior living infant, weight 6 lb. 3 oz.

Case 131. White, para 1, gravida 2, aged thirty-eight, short stout; pelvis android tendency, narrow outlet; anteroposterior 12.25 cm., transverse 13.25 cm. Labor, 20 hr. test then low cervical cesarean section delivery living infant, weight 5 lb. 8 oz.

Case 132. Negro primipara, aged seventeen; contracted gynecoid pelvis; anteroposterior 9.5 cm., transverse 10.75 cm. Labor, 1st stage 15 hr. 30 min., 2nd stage 2 hr. 30 min. Spontaneous delivery living infant, weight 6 lb. 8 oz.

Case 133. White, para 5, gravida 6, aged thirty-six, cardiac patient recently decompensated; large gynecoid pelvis android tendency; anteroposterior 12 cm., transverse 13.5 cm. Labor, medical induction

1st stage 2 hr. 30 min., 2nd stage 35 min. Spontaneous delivery O.L.A. living infant, weight 6 lb. 4 oz.

Case 134. White, para 1, gravida 2, aged twenty-nine; gynecoid pelvis; anteroposterior 11 cm., transverse 13 cm. Outline of head hidden behind pelvic brim. Labor, 20 hr. test, then low cervical cesarean section delivery of living hydrocephalic infant, weight 9 lb. 8 oz., with extremely hard head. The parents refused the operation of choroid cauterization but consented one month later when the head had grown enormously and the operation proved fatal.

Case 135. White primipara, aged eighteen, dystrophia dystocia syndrome with toxemia; gynecoid pelvis; anteroposterior 11 cm., transverse 12.75 cm. Labor, 1st stage 31 hr. 24 min., 2nd stage 7 hr. 24 min. Mid pelvis Kjelland application of Kjelland forceps O.L.T. delivery living infant, weight 8 lb. 15¼ oz.

Case 136. Negro primipara, aged twenty-one, toxemic; contracted pelvis android tendency; anteroposterior 9 cm., transverse 11.25 cm. Labor, 1st stage 20 hr., 2nd stage 2 hr. 30 min. Mid pelvis Kjelland application of Kjelland forceps O.L.T. delivery living infant, weight 7 lb. 15 oz. (delivery required five 45 second tractions at intervals of a minute and a half with measured pull of 115 lb. The infant, uninjured, breathed spontaneously).

Case 137. White primipara, aged nineteen, toxemia with generalized edema; pelvis android tendency with narrow outlet; anteroposterior 11 cm., transverse 12 cm. Labor, 1st stage 23 hr., 2nd stage 4 hr. Mid pelvis Kjelland application of Kjelland forceps O.L.T. delivery living infant, weight 7 lb. ½ oz.

Case 138. Negro primipara, aged sixteen, toxemia; anthropoid pelvis with android tendency, narrow outlet; anteroposterior 11.25 cm., transverse 11 cm. Labor, 1st stage 7 hr., 2nd stage 1 hr. 55 min. Spontaneous delivery direct occiput posterior living infant, weight 6 lb. 12 oz.

CONCLUSIONS

An analysis of these cases shows that while each patient in labor must still be individualized, enough information may be obtained from the single grid film to prognosticate delay definitely due to fetopelvic disproportion. The method's freedom from technical error, its ease of performance by any technician and its economy (a single 8 by 10 inch film) warrant its wider use not only in private practice, but also in the clinic, where its use in all doubtful cases practically doubles the amount of scientific information ordinarily available. Specifically:

(1) If there is a free space 1 cm. wide between the image of the fetal head and that of the pelvic inlet, there should be no pelvic interference with labor unless the iliac spines markedly protrude into the pelvis as in Cases 117 and 130.

(2) Thoms[4] and others have shown that the length of the suboccipitobregmatic diameter is very nearly equal to that of the biparietal, while the occipitofrontal diameter is much longer. Therefore, for practical purposes, when the image of the fetal head approaches the circle, it is to be assumed that the view of the head is that of marked flexion, and one can assume that it is engaged or engaging, while if the image is markedly oval, the head must be extended, and one can assume that it is still floating. A diagnosis of floating head is evident if the borders overlap the pelvic brim in the roentgenogram.

(3) Now if the head is thus seen to float high, one must assume that its image is relatively larger than actual, and this must be taken into account in predicting the outcome of these relatively few cases.

(4) The most important observation made in this study is that if the conjugata vera is 9 cm. or more, a test of labor should be allowed, and in most cases the outcome should be successful. A test of labor ordinarily should include uterine contractions of forty seconds' duration recurring every two to five minutes for twenty to twenty-four hours. At the end of that time, the head should be at least in midpelvis. If not, and if no vaginal examination has been made, it has been shown that the lower cesarean section operation is quite safe.

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SYPHILITIC ULCER ON THE GREATER CURVATURE OF THE STOMACH

REPORT OF A CASE

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IN a paper published in 1931, Sproull[3] gives a critical consideration of 24 cases of gastric ulcer on the greater curvature of the stomach described by David and 3 cases described by Blaine. He comes to the following conclusions:

1. The roentgenological diagnosis of a benign peptic ulcer on the greater curvature is not possible.

2. Many ulcers are described as located on the greater curvature although they are situated either on the anterior or posterior wall.

3. The diagnosis can be absolutely certain only when confirmed by histological examination.

4. Every alteration in the contour of the greater curvature must be considered as suspicious of cancer and a dietary treatment not permitted.

Only in the case of syphilis does he make an exception.

As to his conclusions, it seems that others have also to deal with these difficulties and have looked for typical signs by which it may be possible to differentiate between a benign ulcer and a malignant lesion.

Gutzeit and Kuhlmann[1] published their findings on this subject. They describe two signs which should be typical for an ulcer on the greater curvature: (1) an overlapping of the ulcer by the surrounding mucosal folds which gives a double contour on the film; (2) a transverse fold arising from the ulcer and crossing the normal longitudinal folds. The first sign should be seen only in recent ulcers, while the second should appear in ulcers of longer standing. According to these authors the presence of the signs depends on the greater or lesser fixation of the mucosa to the under layer and the elasticity of the mucosa itself. Infiltration of the mucosa would thus exclude the signs.

Among the patients who came to our clinic in 1938 there was one case which we diagnosed as an ulcer on the greater curvature which turned out to be an error.

Fig. 1

The patient, G. M., female, married, came to our clinic on February 16, 1938, because of gastric pain from which she had suffered since October, 1937. The pains were localized in the upper part of the abdomen and sometimes in the back. They appeared in attacks and were very violent. They had no relation to meals. During the two previous months the attacks had grown worse so that she now suffered the whole day and night. She often vomited a mucous fluid, never acid or blood. No pyrosis. She

felt very weak and had lost weight. Defecation was regular once a day; the feces had never looked black. Micturation undisturbed. Menstrual history irrelevant. On general examination no abnormalities were found in the heart and lungs; pulse 72, blood pressure 105/70. In the epigastrium there was a tender spot, with a slight muscle spasm. No abnormal findings in the blood or urine. The Wassermann test was not done. The benzidine test was negative. In the fasting gastric juice there was no free hydrochloric acid or other acids. A few days after the first examination there seemed to be a palpable tumor to the left above the umbilicus. With a tentative diagnosis of tumor the patient was sent to us for roentgenological examination.

On roentgenoscopic examination the stomach appeared rather small. On the greater curvature appeared a large niche. From the cardia and lesser curvature two wide mucosal folds extended to the niche. A very large one crossed the whole width and seemed to divide the stomach in two parts. At the end of this fold there were two circular light spots (Fig. 1). We did not see evident peristaltic waves and palpation showed a rather slight rigidity. The passage was free through the entire stomach. After four hours the stomach was empty, except for a small quantity of barium in the niche.

With regard to the niche and the transverse folds as we knew them from the publication of Gutzeit and Kuhlmann we made a diagnosis of gastric ulcer on the greater curvature and advised operation. This was performed. A gastric resection (Billroth II) was carried out. During operation the surgeon could not discover any deformity on the outside of the stomach except a slight rigidity. After operation the Wassermann reaction was 2 plus. The patient was submitted to an antisyphilitic treatment and made an uninterrupted recovery. We kept her under opservation for nine months and she is still in perfect condition.

The pathologic examination of the resected part showed ulcer of the mucosa on the greater curvature with infiltrated tissue all around it. On the oral side there was a wide mucosal fold. Microscopic examination showed inflammatory infiltration of the mucosa with leukocytes and plasma cells. Syphilis (?).

When we consider the roentgenological findings in our case with the peculiarities Gutzeit and Kuhlmann observed in theirs,

Fig. 2

it seems that the same irregularities can be found in syphilitic ulcers with strongly infiltrated mucosa. They explain these folds as due to the loose attachment of the mucous membrane to the deeper layers and the less tense condition of the mucosa. But considering our case, it seems to me that a loose attachment cannot be the sole cause as we saw the same peculiarities in a strongly infiltrated mucosa. Moreover, the signs Gutzeit and Kuhlmann called attention to may be absent in histologically confirmed peptic ulcers on the greater curvature (see Van Buchem).

The reason that we did not consider the possibility of syphilis may be the fact that syphilis of the stomach is extremely rare in this country. This is a single case in three years with an average of 1,600 gastrointestinal examinations a year. Finally, we come to the conclusion that the roentgen signs of Gutzeit and Kuhlmann are not characteristic of all ulcers of the greater curvature.

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SOME PHYSIOLOGICAL CHANGES IN THE SKIN PRODUCED BY NEUTRONS*

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THIS study of the response of the skin and its minute vessels to neutron irradiation was undertaken to elucidate some of the physiological changes in the skin produced by neutron irradiation, though the exact chemical nature of these changes is unknown. This in turn may lead to an understanding of some effects of irradiation in normal and cancer tissue and is, therefore, of considerable importance to the radiologist. Lewis and Zotterman[5] studied the effect of ultraviolet irradiation on skin capillaries, but to date the effect of irradiation used in cancer therapy on the minute vessels of the skin has never been reported. The neutron radiation was produced by the 60 inch Berkeley cyclotron. The skin is a most available structure, is easy to observe from day to day, and quite accessible for injecting chemical substances. The physiological reactions here include vasodilatation and vasoconstriction of the capillaries, changes in their sensitivity produced by the irradiation, changes produced by injecting chemical substances into the skin itself, and skin temperatures.

When the skin is subjected to injury its response is much the same regardless of the nature of the injury or the manner in which it is produced. Injuries may be produced by heat, cold, inflammation of bacterial origin, the Faraday current, the injection of chemical substances, a blow from an object, ultraviolet rays, roentgen rays or neutrons. Whether or not reddening, the erythema, appears depends only on the state of the minute vessels of the skin. If some refractive oil is placed on the skin and then illuminated with an intense light and observed with a binocular microscope, the minute vessels can be seen when dilated. One should also filter out the infra-red rays to prevent undesired reactions resulting from the heat absorbed. Under normal conditions the capillaries are not observed but if the skin is stimulated, causing the vessels to dilate, they are seen standing up perpendicular to the skin, running towards the surface and returning to the deeper structures, forming a loop. At the base of the loop one sees an extensive network of vessels, the subpapillary plexus, which are of the same caliber but somewhat deeper in the skin. At a glance it is evident that the presence or absence of these dilated capillaries determines whether or not the skin appears erythematous. The red color of the skin is mostly due to the vessels in the subpapillary plexus, as they are ten times more numerous than the capillary loops.

While it is true that the erythema will appear during the time the skin is reacting to injury, there is a great variation in the time of its appearance and also in the time required for healing to take place. In the case of erythemas produced by heat and cold, a scratch, or a mechanical blow, the reddening will be seen in a few seconds. Those produced by the ultraviolet rays appear in a few hours, and those with roentgen rays in a few weeks.

METHODS FOR STUDYING THE ERYTHEMA PRODUCING VASODILATATION AND RELATIVE PHENOMENA

The drawing of a pointed object over the surface of the skin is called the scratch test, and in a normal individual causes a definite series of events to take place. There is first the so-called white reaction appearing as a white line where the stimulus has been given. It is caused by the mechanical

* The neutron therapy is aided by a grant from the National Cancer Institute.

stretching of the walls of the endothelial tubules in the skin. This is a protective mechanism for preventing injurious dilatation of capillaries, causing them to constrict once they are stretched by the blood in their lumina beyond a certain limit.

If a very light stimulus is given this may be the sole reaction; however, as a rule a red line extending somewhat beyond the limits of the white reaction soon replaces it. This so-called red reaction results from the liberation of a chemical substance generally believed to be similar to the histamine formed by injured cells. Irradiation may also injure cells and cause the formation of a histamine-like substance (Ellinger[2]), which in turn causes vasodilatation and the appearance of the erythema. Holtz,[3] using ultraviolet light, irradiated solutions of the amino acid histidine, and actually obtained very small quantities of the vasodilative substance histamine. Histidine has been found in high concentrations in the skin by Jono.[4] It is only necessary for the irradiation to cause decarboxilization of histidine to get histamine, as was suggested by Ellinger.[1]

Later a more diffuse reddening, called the flare, is seen just outside this red reaction, extending out many millimeters or even centimeters. The local color of the red reaction is due to dilatation of the minute vessels, and the flare to a dilatation of the arterioles. The flare is called forth by a nervous mechanism.

When one presses firmly on making the scratch test, or even lightly on persons with factitious urticaria, there will appear still another reaction. In place of the white reaction there will appear a narrow band of local edema, causing an elevation of several millimeters in the skin. This edema results from an outpouring of fluid by the minute vessels into the interstitial spaces, causing pressure on these minute vessels and their collapse. This makes the skin in the narrow edematous line appear pale. The transuding fluid soon diffuses into the surrounding tissues and the wheal fades. The wheal appears only in the precise area where the red reaction will appear.

If one places a drop of 1:300 histamine solution on the skin of a normal individual and pricks the skin through it, there appears a reddened area and soon afterwards a raised edematous wheal is formed in its center. If only a small amount of histamine is used only the red reaction will be seen, indicating dilatation only of the minute vessels, but if a larger amount is used there will be in addition a pouring out of fluid into the interstitial spaces and the formation of the wheal. On the other hand, if one uses a drop of adrenalin or pituitary extract on the skin there will be instead a constriction of the minute vessels. Histamine is much more powerful as a vasodilator than adrenalin and pituitrin are as vasoconstrictors and can easily overcome their pharmacological actions.

This paper is an application of this well known physiology and pharmacology, discussed in great detail by Sir Thomas Lewis, to the special case of the effects of neutrons on the capillaries of the skin. This work was carried out during the past six months on cancer patients being treated with neutrons at the Crocker Radiation Laboratory of the University of California at Berkeley.

APPLICATION OF TESTS TO IRRADIATED AREAS

An erythematous area is selected, preferably one where there is considerable soft tissue so that the walls of the minute vessels may be easily stretched. The gluteal fields of patients who are being treated for cancer of the prostate have proved ideal. A pointed instrument is drawn through the irradiated area and on into the non-irradiated area, the latter serving as a control. Immediately a white line will be seen extending throughout the erythematous and control regions. After a few seconds the red reaction appears in the control, but the white reaction may persist in the erythematous skin until all vestige of it disappears in the control area (Fig. 1). On occasions when the erythema is slight it is possible to determine the edges of the treated field by this scratch test. A rapid transition from white line to red

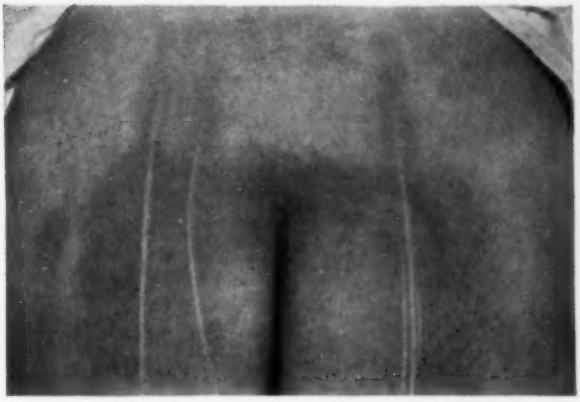

Fig. 1. (*top*) Usual scratch test seen running through erythematous area. The scratch test on the extreme left was made immediately before the picture was taken and shows a marked white reaction in both the control and irradiated areas, not sufficient time having elapsed for the later reactions to take place. There is also a flare seen in all control areas.

The two mesial scratch tests were made thirteen and fifteen minutes before the lateral test. They all show a persistence of the white reaction in the irradiated areas. There is a complete disappearance of the white reaction in the control area of the mesial tests, the white reaction being completely replaced by the red reaction. In the lateral test the white reaction can be seen in the control areas.

Fig. 2. (*bottom*) Scratch tests in patient with factitious urticaria. The lateral test was made fifteen minutes before the picture was taken. In it the early reactions have disappeared in the irradiated area, but the wheal is still present in the control area. There is no flare at this time. The reddening on the mesial side appeared only after the flare was formed in the other test.

The mesial test was made just before the picture was taken. It also has a wheal in the control area, but the white reaction is still present in the irradiated area to show the area has also been stimulated. There is a striking difference between the white reaction and the edematous wheal. The flare is seen in both the control and irradiated areas, though it is less marked in the latter.

line is seen at the junction of the irradiated and non-irradiated fields. This indicates a change in the response of the minute vessels in the irradiated area. From this one may conclude irradiation either increases the sensitivity of the walls of the endothelial tubules to the scratch test, bringing about the abnormal white reaction, or else there is an overcoming of the histamine reaction so that vasoconstriction persists. One must of course assume here that vasodilative histamine-like substances are antagonistic to the vasoconstrictive mechanism, and whether there is vasoconstriction or vasodilatation is determined by which effect dominates. This overcoming of the histamine reaction may be due then to failure of it to form, or its actual destruction, or unresponsiveness of the irradiated area to it.

An even more striking reaction may be produced by this simple scratch test if one is fortunate enough to find a person so sensitive to it that wheal formation is easily produced. We treated a seventeen year old boy with an osteogenic sarcoma of the hip who was found to be extremely sensitive to the scratch test. On drawing a pointed object over his erythematous fields one saw immediately a white reaction extending through the erythematous and control areas. In a few seconds there was a red reaction in the control area and to a lesser degree in the irradiated part. The wheal developed in a few minutes in the control but usually no wheal formation was to be seen in the erythematous area. Between the two regions an actual hump produced by the edema on the control side could be felt (Fig. 2). One must conclude that in the erythematous area histamine causes no change in the permeability of the capillary walls and no transudation of fluid into the interstitial spaces, and hence no local edema. This failure of wheal formation indicates absence of histamine action, and cannot be found during a course of protracted therapy until the erythema appears.

On many occasions on carrying out the scratch test, minute pieces of the epidermis are elevated from the cutis and on being isolated from the minute vessels appear white, giving much the same appearance as the white reaction. This is not to be confused with the purely vascular reaction discussed here.

In both the irradiated and control areas the flare can be seen in the early part of the scratch test, extending out several centimeters from the path of the scratch test. From this one may conclude that the nervous mechanism controlling flare formation is not affected by amounts of radiation sufficiently great to produce an erythematous reaction.

To prove that these areas are no longer sensitive to histamine, it is only necessary to place histamine 1:300 on the skin and prick it into the subcutaneous tissue and at the same time into a neighboring area outside the irradiated field. In the control area there will be a typical wheal formation with a flare, but even with this high concentration of histamine there is no evidence of wheal formation in the irradiated area. The flare, of course, cannot be distinguished in the already reddened field. It was first thought the reason for the irradiated area being refractory to histamine was due to dilatation of vessels and the congestion inhibiting wheal formation mentioned earlier. It was thought that the dilated vessels permitted the injected histamine to diffuse away to such an extent that it was no longer sufficiently concentrated to bring about wheal formation and to prevent the formation of a pressure gradient in the minute vessels. This was proved not to be true by first injecting a solution of 1:1000 adrenalin, causing a vasoconstriction so that when the histamine was injected it would remain localized. One would expect, since the histamine is a more powerful vasodilator than the adrenalin, a vasoconstrictor to have wheal formation; but this was not found to be the case, and there was a rapid disappearance of the blanched area without wheal formation. This is additional evidence that these areas are re-

fractory to histamine and also that congestion cannot explain the failure of wheal formation. This contraction of the minute vessels in an erythematous area with the injection of adrenalin also indicates that the vessels are still able to function in spite of the irradiation effects.

SKIN TEMPERATURES AND VASODILATATION

To investigate further and to attempt to show that vasodilatation takes place only a few days before the erythema is seen during a course of protracted neutron therapy, skin temperatures were measured. If there is vasoconstriction or vasodilatation there will be a change in the skin temperature, and it is only necessary to measure this temperature to tell which is present. This can be done accurately and rapidly by the use of thermocouples (Fig. 3). Two strips of silver about 1 cm. square are connected by Constantan wire, and two copper wires connect the silver plates to the terminals of a very sensitive galvanometer, a Leeds and Northrup Type R. If a temperature gradient exists between the plates there will be a greater flow of electrons from the warmer plate, resulting in a difference in potential measured by the deflection of the galvanometer. By using an ice bath for a standard and comparing it to another known temperature such as an alcohol-ice bath, the galvanometer and thermocouples can be calibrated. Since a deflection of 3.25 cm. was produced on the galvanometer with a known difference of temperature of 1° Centigrade, temperature differences of 0.1° were considered reliable.

It was necessary to take precautions to make reproducible measurements. If this thermocouple is held against the skin too firmly or too long there will be a mechanical pressing of blood from the area, and a fall in temperature will result. It is then necessary to alternately apply and remove the thermocouples at definite intervals every few seconds; thereby consistent and reproducible skin temperature measurements are obtained. It is also necessary to avoid pressure on the area during irradiation, such as might be produced by pressing against the cone of the therapy unit, as the external pressure affects the blood supply to the area. One will also often find abnormally low skin temperatures on attempting to measure temperatures directly over large metastatic glands. The tension in the skin

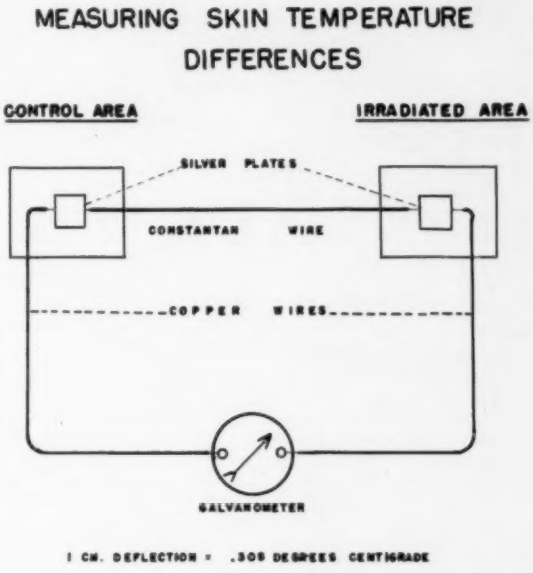

Fig. 3

caused by the mass will reduce the blood supply to the minute vessels and result in a similar cooling.

When these precautions are taken one will find during the first eight to ten days immediately after each fraction of treatment a slight cooling of the irradiated area not existing before the treatment. This cooling is followed by a partial return to normal temperature during the next forty-eight hours and depends on the amount and rapidity of treatment. After approximately twelve to fourteen days there is little difference in the temperature of irradiated and non-irradiated areas before treatment, and one can now expect the erythema to appear in a few days. Once the erythema develops there is a steady increase in temperature in the irradiated area, which is now warmer than the control area, though for some time there may be a slight fall in temperature

immediately after treatment. This fluctuation before and after treatment and the changes throughout the entire course of irradiation are recorded by the heavy curve of Figure 4.

Finally, several days before the epidermolysis is seen there will be no difference in the skin temperature before and after treatment, and it remains elevated at all times. This elevation may amount to as much as 3° C. Thus until the beginning of the erythema there is no vasodilatation and no active histamine-like substance, and there may be actual cooling and vasoconstriction. When there has been an actual fall in temperature there is probably a destruction of the normal vasodilatation or an injury to the accompanying vasodilative mechanism. After the erythema begins, vasodilatation can, of course, be seen (Löw-Beer and Redisch[7]), but there is no pharmacological action of histamine, as the area is now refractory to its action even when high concentrations are injected into the skin.

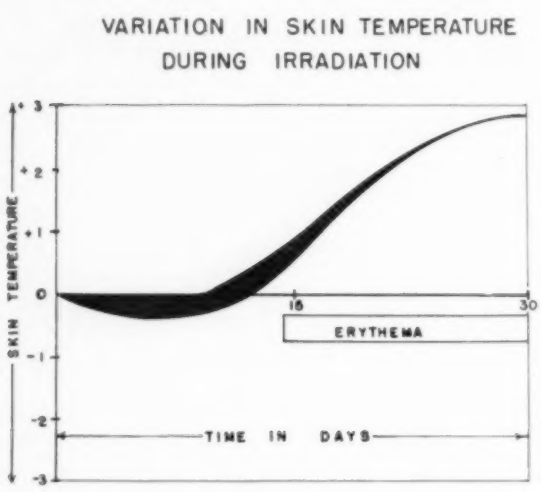

FIG. 4

It has been of some interest to study skin temperatures on patients returning for check-up examinations. It might be expected that the minute vessels would be injured by the irradiation. In a number of cases skin temperatures were measured and not a single case could be found that showed the slightest difference between the non-irradiated and the irradiated areas. This would suggest an adequate revascularization. Pathologists have long described the vascular changes in irradiated areas (Windholz[8]), but they apparently have no effect on the temperature of the skin, which depends on the minute vessels.

SUMMARY AND COMMENTS

The scratch test in erythematous areas shows a persistent white reaction compared to control areas, indicating an increased sensitivity of the endothelial tubules.

The scratch test in erythematous areas in individuals with factitious urticaria shows little or no wheal formation. This indicates that the erythematous skin is refractory to histamine action.

On injecting histamine 1:300 into erythematous fields there is no wheal formation. The failure of wheal formation in erythematous skin is then apparently not due to the absence of histamine but to the refractoriness of the area. This failure cannot be due to just congestion, as on injecting vasoconstrictive substances to obliterate it there is still no wheal formation.

Vasodilative substances are not formed during the latent period before the erythema is seen, as there is no elevation of skin temperature measured by the thermocouples.

There is a marked rise of skin temperature indicating vasodilatation after the erythema is seen.

The mechanism responsible for constriction of minute vessels in erythematous areas is still sensitive to vasoconstrictive substances in spite of the erythematous reaction, as is shown by the blanched areas after adrenalin is injected. The dilatation of the minute vessels is then a reversible reaction.

Vascular changes produced by obliteration of arteries and veins seen in pathological material in post-irradiation areas have no definite effect on skin temperature and thus on the minute vessels. This sug-

gests an adequate revascularization of the area.

The nervous mechanism controlling flare formation is not affected by irradiation.

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FIBROSIS OF THE LUNG FOLLOWING ROENTGEN IRRADIATION FOR CANCER OF THE BREAST*

A CLINICAL STUDY

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RADIATION pneumonitis is familiar to most radiologists as a fairly common sequel to the irradiation of extrinsic and intrinsic thoracic tumors. Since 1922, when the condition was introduced into American medical literature by Groover, Christie and Merritt,[8] pulmonary fibrosis has received considerable attention and has been the subject of numerous studies. The most recent are those of McIntosh and Spitz,[24] Freid and Goldberg,[6] Warren and Spencer,[31] and Jacobsen.[19] Since these authors have reviewed and discussed the literature, it seems unnecessary to do so at this time.

The material for the present study consists of cases of breast cancer treated on the service of Dr. Frank E. Adair at the Memorial Hospital. We have endeavored to correlate the various physical factors of irradiation with the frequency of pulmonic complications and to emphasize certain physiological disturbances which are considered important, and to some extent capable of relief with the aid of certain measures.

TECHNIQUE AND FREQUENCY

In order to determine the frequency of this condition, we have reviewed the course of patients with operable cancer, treated partly or entirely by irradiation between 1926 and 1936, inclusive, and who are still alive. This five year or longer interval should eliminate any error relative to the differential diagnosis of pulmonary fibrosis and metastases. There were altogether 347 cases, of which 77 (22.1 per cent) developed roentgenographic evidence of pulmonary fibrosis. This number is presumed to be slightly inaccurate because postoperative roentgenographic studies were made only in the presence of symptoms.

During this time, there were changes in the technique of irradiating the breast and the axilla. Massive doses were replaced by divided doses, the use of interstitial radium was largely discontinued and the time of treatment relative to that of the operation was altered. Other factors, such as the areas irradiated, the position of the patient relative to the beam of radiation, the skin target distance and the voltage, were less radically changed. Hence, before a further analysis is made, a brief description of the more constant factors will be given.

In irradiating the breast, treatments were usually given through two portals. First, a medial portal included the region from the parasternal line to the nipple and from the third rib down to the sixth or seventh rib. Second, a lateral field covered the area from the nipple to the anterior axillary line. The beam was directed perpendicularly when irradiating the medial field but tangentially for the lateral. The axilla was treated through three portals. There was an anterior axillary portal which was roughly rectangular and extended from the base of the neck to the shoulder joint and included the supra- and infraclavicular regions as well as a portion of the upper anterior chest. The posterior axillary portal covered an equal or slightly larger area and extended from the upper dorsal vertebra to the outer margin of the scapula. The direct axillary field was square and included the entire lateral surface of the axilla. The beam was directed straight across the chest when

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treating the anterior and posterior axillary fields but slightly upward when irradiating the direct axillary portal. In all cases, unless otherwise specified, 200 kv., 30 ma., filtered with 0.5 mm. copper plus 2 mm. aluminum were used. The skin target distance was 50 cm. routinely, except occasionally when 70 cm. was used for the direct axillary field.

TABLE I

FREQUENCY OF PULMONARY FIBROSIS FOLLOWING VARIOUS TECHNIQUES OF IRRADIATION

Technique	Number of Cases	Pulmonary Fibrosis
I. Preoperative		
1. Massive doses (500–750 r per portal)	11	1
2. Divided doses (300 r ×6 = 1,800 r per portal)	57	31
3. Radium element pack (8,000 ×3 = 24,000 mg-hr. per portal)	31	2
II. Postoperative		
1. Massive doses	77	7
2. Divided doses	41	8
3. Radium element pack	7	0
III. Pre- and postoperative		
1. Massive doses	37	8
2. Divided doses	3	1
IV. Miscellaneous		
1. Massive doses, interstitial radium, surgery	40	3
2. Divided doses, interstitial radium, surgery	19	8
3. Interstitial radium, surgery	8	1
V. Irradiation only	16	7
TOTAL	347	77

In determining the frequency of pulmonary fibrosis, the patients have been grouped with regard to the time of operation and the technique of irradiation (see Table 1).

Preoperative. Ninety-seven cases received preoperative irradiation, and of these, 11 were treated with single doses of 500 to 850 r (measured in air). In most cases only one treatment per field was given. In this group there was one case of pulmonary fibrosis. Fifty-seven cases were treated with divided doses. Three hundred roentgens were given to two fields daily with the axillary portal treated on a separate day. The total varied from 1,200 to 2,400 r, averaging 1,800 r per portal in most instances. Of 57 patients treated in this manner, there were 31 who suffered from pulmonary fibrosis. Thirty-one patients were treated with the 4 gram radium element pack and, although an equivalent skin reaction was obtained, only 2 developed pulmonary changes.

Postoperative. In treating the postoperative cases, high voltage roentgen therapy was used on the axillary portals, while low or intermediate voltage was used in treating the operative fields. Seventy-seven patients were treated with massive doses to all five fields, usually two cycles of 750 r each, separated by an interval of about three months. In this group there were 7 cases of pulmonary fibrosis, but where divided doses were used in 41 cases, 8 patients developed this complication.

Preoperative and Postoperative. When both pre- and postoperative roentgen therapy was given in massive doses to 37 cases, 8 developed fibrosis of the lung.

Miscellaneous. There was a miscellaneous group of 67 cases in which roentgen therapy (usually massive doses), interstitial radium and surgery were used. In this group 12 cases of pulmonary fibrosis were found. Eight of these were associated with the use of divided doses of radiation.

Irradiation Alone. Of the 16 patients with breast cancer treated by irradiation alone, 7 developed fibrosis of the lung.

AGE

The question of age has received considerable attention and many agree that, other factors being equal, elderly people are more prone to develop pulmonary fibrosis. This seems reasonable and yet the average age of the 77 patients was 45.6 years and the mean age was forty-six. Furthermore, some of our most severe cases have been

noted in individuals in their thirties. These patients had not received a comparatively larger amount of radiation.

DEPTH DOSE

In investigating doses received by the lung tissue, one meets with many difficulties. The depth doses cannot be accurately correlated with the symptoms, roentgenographic, and physical findings. One must consider the inherent differences in tissue reaction to irradiation, variations in the size of the breast and its location relative to the thorax, as well as the exact direction of the beam of radiation. We have therefore limited our calculation to the axillary region, where the anatomical structures bear a fairly constant relationship and where there is less variation in the size of the fields. It was believed that when our technique was used, the region of maximum intensity in the lung was located directly beneath the point where the mid-clavicular line crossed the second rib. Measurements of this region were taken with calipers. From these figures depth doses in terms of threshold erythema doses (T.E.D.) were estimated in 15 representative cases of pulmonary fibrosis. The depth dose varied from 2.5 to 7.3 T.E.D., averaging 5.2 T.E.D. for the 15 cases.

The threshold erythema dose as defined by Quimby[27] is the amount of radiation which produces a faint reaction in 80 per cent of all cases, the remaining 20 per cent showing no visible reaction. For 200 kv. roentgen rays this means about 650 r measured on the skin, including backscatter. Hence, the doses specified above are from about 1,600 to 5,000 r to the lung, averaging about 3,500 r.

PATHOLOGY

To date, no fully adequate pathological study of the post-irradiation changes in human lungs has appeared. However, difficult as it may be to interpret the pathological alterations seen in some lungs after irradiation, no reasonable doubt can remain that some of these changes are sequelae of the action of roentgen rays. How specific these findings may be is not a matter of complete agreement among various observers at the present time.

On clinical and pathological evidence, it is known that post-irradiation pleuropulmonitis may be a transient reaction in lungs and may disappear, leaving no demonstrable residue. On the other hand, it may not subside but persist, eventually going on to produce permanent fibrotic lesions. These intrapulmonary lesions are commonly considered to be the cause of the clinical symptoms observed by the patient. That they are causal factors cannot be denied, but we question whether or not they play the major rôle.

The changes following irradiation are analogous fundamentally to those that occur in other organs, differing only as these organs vary among themselves in their tissue organization. Inasmuch as the lung substance can only be irradiated through the parietes, it is expected that the overlying skin may share in the ill effects produced, just as skin is apt to do in any field of irradiation. Particularly in mammary cancer, large skin areas receive radiation and some portion of these develop late radiation changes characterized by endarteritis, thrombosis, loss of elastica and skin appendages, with replacement fibrosis with or without surface ulceration. Thus, a normally pliable skin and subcutaneous tissue may be made stiff and inelastic. These changes are not necessarily limited to this portion of the chest wall but in severe cases may involve the underlying muscle and rib.

CLINICAL OBSERVATIONS

This study is limited to the chronic type of irradiation fibrosis. Often insidious in its onset up to many months after treatment for the original neoplastic disease, the chief symptoms are persistent, slightly productive cough, dyspnea on exertion and fatigue. Hemoptysis, pain, and palpitation are less common.

Classically, deviation of the trachea, retraction of the mediastinum to the affected side, diminished thoracic expansion, im-

paired percussion note, and high pitched tubular breath sounds are easily recognized. Less intense forms of the disease often escape detection, although they may cause marked distress to the patient.

It must be emphasized at this point that we have been unable to determine any significant correlation between the symptoms, signs, degree of fibrosis and amount of radiation to the lung. While it is true in general that the more roentgens delivered, the more severe the late pulmonary symptoms are likely to be, outstanding exceptions are so frequent that one cannot make it a clinical axiom.

The claim has been made that right-sided heart failure may result from irradiation fibrosis.[31] During the past three years at the Memorial Hospital, we have not observed one instance of chronic cor pulmonale in patients with uncomplicated irradiation fibrosis. Routine roentgenoscopy of patients has failed to show any enlargement of the right ventricle or pulmonary conus. However, when the mediastinum is markedly displaced and fibrotic, right heart failure may occur not because of the pulmonary scarring, but because of angulation of the pulmonary vessels with resultant right ventricular strain, hypertrophy, and finally dilatation. Consistent changes in the electrocardiogram are observed very frequently but it is now known[23] that these are not the result of primary or secondary cardiac disease but are due to alterations in the relationship of the heart to other structures in the chest.

PHYSIOLOGICAL STUDIES

The mechanism of the dyspnea in these patients has not been explained to date.

TABLE II

PLASMA CO_2 CONTENT AND COMBINING POWER AND BLOOD PH VALUES IN A GROUP OF PATIENTS WITH IRRADIATION PULMONARY FIBROSIS BEFORE, IMMEDIATELY AFTER AND TEN MINUTES AFTER EXERCISE. SEE DISCUSSION IN THE TEXT

Patient	Age	CO_2 Plasma Content			CO_2 Combining Power			pH Blood		
		Rest	Immediately after Exercise	10 min. after Exercise	Rest	Immediately after Exercise	10 min. after Exercise	Rest	Immediately after Exercise	10 min. after Exercise
B.C.	58	69	57	69				7.79	7.77	7.81
E.B.	51	69	65	65	62	58	54	7.84	7.92	7.97
N.B.	43	65	62	56	63	60	59	7.56	7.56	7.62
F.G.	47	64	56	59	66	58	62	7.57	7.74	7.70
C.G.	41	67	60	63	66	60	61	7.64	7.62	7.63
I.D.	62	66	61	61	71	62	62	7.81	7.80	7.92
S.S.	41	69	66	69	70	66	67	7.77	7.77	7.80
R.D.	43	70	69	67	75	70	65	7.85	7.85	7.80
M.M.	61	71	66	66	71	66	67	7.80	7.89	7.92
G.Z.	48	60	59	55	62	59	60	7.61	7.61	7.62

The cause of the cough is obvious since pathologically there is a persistent chronic bronchitis in the affected area.

Since a complete understanding of the dyspnea is necessary for application of the principles of the treatment we advocate, one must consider the mechanism of dyspnea.

There are two theories extant at present explaining shortness of breath due to muscular effort. One, subscribed to by Haldane, Gesell, Howell and others,[18,25,32] is that stimulation of the respiratory center is caused by (*a*) an increase in the CO_2 plasma content, and (*b*) an increase in the hydrogen ion concentration in the respiratory center. Theoretically, muscular exercise causes this.

TABLE III

PLASMA CO_2 CONTENT AND COMBINING POWER, BLOOD PH VALUES IN A GROUP OF CONTROLS BEFORE, IMMEDIATELY AFTER AND TEN MINUTES AFTER THE SAME EXERCISE THAT WAS PERFORMED BY THE PATIENTS

Patient	Age	CO_2 Plasma Content			CO_2 Combining Power			pH Blood		
		Rest	Immediately after Exercise	10 min. after Exercise	Rest	Immediately after Exercise	10 min. after Exercise	Rest	Immediately after Exercise	10 min. after Exercise
J.F.	33	65	64	60	70	63	61	7.66	7.55	7.56
F.F.	29	71	68	65	71	66	64	7.57	7.58	7.58
J.L.	32	69	62	60	64	54	62	7.89	7.83	7.58
M.M.	26	67	64	65	68	60	62	7.74	7.76	7.72
L.R.	50	57	60	58	61	61	57	7.77	7.90	7.80
C.H.	40	67	67	65	64	60	67	7.58	7.56	7.51

The other theory, formulated by Peabody[26,29] and later developed by Harrison and his co-workers,[7,11–16] and others, is that dyspnea secondary to muscular activity is due to increased ventilation caused by activation of the respiratory center, not by humoral agents but by nervous stimuli arising in the muscles and carried to the medulla by way of the spinal cord. They found that dyspnea is directly proportional to the ventilation and inversely proportional to the vital capacity. (Roughly, the maximum possible ventilation per minute equals 25 times the vital capacity.)

In these patients with a partially nonfunctioning fibrotic lung, it seemed that the chemical theory of dyspnea might apply. Blood samples taken before, immediately after and ten minutes after exercise and analyzed for CO_2 content, and pH values, showed consistently lower CO_2 values and essentially unaltered or higher pH readings after exercise (Table II).

The same CO_2 and pH changes occurred with a group of normal controls after excercise, but were less marked (Table III).

Obviously, Haldane's theory did not obtain. From these studies it was further sug-

TABLE IV

SHOWING THE VITAL CAPACITIES OF 12 PATIENTS WITH VARYING DEGREES OF RADIATION FIBROSIS. NOTE THAT PATIENT F. G. WITH GRADE 2 FIBROSIS HAS A VITAL CAPACITY OF 2.5 LITERS WHILE M. G. WITH GRADE 1 FIBROSIS HAS A VITAL CAPACITY OF ONLY 1.7 LITERS

Patient	Age	Sex	Vital Capacity		Evident Fibrosis on Roentgenogram
			Expected	Actual	
B.C.	58	F	3.0	1.7	Grade 1
E.B.	51	F	3.1	2.2	Grade 2
A.G.	67	F	2.9	1.0	Grade 3
M.G.	55	F	3.6	1.7	Grade 1
N.B.	43	F	3.6	2.0	Grade 1
F.G.	44	F	3.7	2.5	Grade 2
C.G.	41	F	2.7	1.6	Grade 1
I.D.	59	F	3.0	1.7	Grade 1
S.S.	52	F	2.7	1.5	Grade 3
R.D.	43	F	3.0	1.2	Grade 3
M.M.	58	F	3.4	1.5	Grade 2
C.Z.	48	F	2.7	1.8	Grade 1

gested that the altered CO_2 and pH values were not the cause of, but were the result of the hyperventilation.

We also found that the vital capacities of these patients are consistently lowered and may be decreased as much as 60 per cent. The decrease of the vital capacity was not a function of the roentgen evidence alone of fibrosis and was much lower than one could account for by the volume

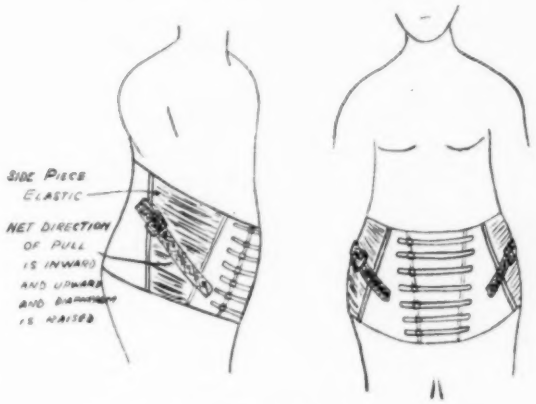

Fig. 1. Diagram of Kerr-Lagen belt.

of pulmonary tissue apparently affected (Table IV).

Since dyspnea is inversely proportional to the vital capacity, it is important to realize the various agents lowering vital capacity in these patients.

From our observations we have found that there are four main factors that cause reduction of the functioning pulmonary capacity in patients with chronic irradiation fibrosis. They are (*a*) fibrosis of the lung; (*b*) a variable degree of fixation of the chest wall; (*c*) compensatory emphysema in the undiseased lung; (*d*) hyperirritability of the Hering-Breuer reflexes in the fibrotic pulmonary tissue.

(*a*) The fibrosis of the lung reduces the vital capacity by subtraction from the total pulmonary volume.

(*b*) The degree of immobility of the chest wall varies with the intensity of the late fibrosis of the thoracic parietes. Not a little of the immobility may be produced by a firm, fibrotic mastectomy scar.

(*c*) With the contraction of the fibrotic portion of the lung, the remaining pulmonary tissue undergoes compensatory emphysema. This is an extremely variable factor and is inversely proportional in part to the retraction of the chest wall.

(*d*) We believe that, since a portion of the lung is fibrotic and distorted, the normal inspiratory and expiratory pulmonary reflexes of Hering and Breuer are more irritable and seriously alter the normal rhythmic action of the entire sequence of pulmonary reflexes.

There is a very rapid normal reduction in vital capacity in patients over fifty-five years. It is quite natural that the more elderly the individual, the greater will be the dyspnea and secondary cough as compared with a younger patient with the same degree of pulmonary fibrosis.

TREATMENT

So far as is known at present, there are no agents that will favorably affect the already diseased lung or diminish the fixity of the chest wall.

We have applied the principle of using an abdominal belt, the function of which is to raise the diaphragm by abdominal pressure.[22] It is comfortable, worn while the patient is up and about and, on the basis of clinical and laboratory data at hand, produces its beneficial effect in one or both of the following ways: (1) In some patients there is an actual increase in the vital capacity when the belt is worn. Careful measurements made on roentgenograms taken at the end of inspiration and expiration with and without the belt show a greater diaphragmatic excursion when the belt is worn. This increase is not at the expense of transverse or sagittal thoracic expansion. While the increase in vital capacity may be small (200 to 400 cc.), computation of the minute ventilation decrease when the belt is worn may be as great as 5 to 10 liters. It may well make the difference between severe respiratory distress and comparative comfort. (2) The belt forces the patient to use the lower, larger

portion of the chest for respiration and puts the upper, more diseased portion of the lung at relative pulmonary rest. Consequently, the irritable Hering-Breuer reflexes arising in the diseased lung are reduced to a minimum and give the patient an increased amount of pulmonary comfort by increasing the tidal air.

Spirographic studies done on these patients with and without the belt after the same exercise show either (1) a decrease of ventilation, (2) an increase in tidal air with the same ventilation, or (3) a decrease in the time of the expiratory phase of the respiratory cycle, or (4) combinations of (1) and (3) or (2) and (3) when the belt is worn.

Weight reduction is the second important phase of the treatment. The great majority of our patients are many pounds over their ideal weight, based on height and age. We have found that judicious dietary restriction with gradual reduction of the weight is enough in many instances to produce a complete remission of symptoms. This is an application of a very well known principle.

We have observed that the severity of the cough is directly related to the degree of the dyspnea. Until the shortness of breath is relieved by one or both of the above measures, mild sedative cough mixtures are used. In well over 90 per cent of our patients the cough has decreased as the dyspnea improves.

CONCLUSIONS

1. In the treatment of cancer of the breast in part or entirely by high voltage roentgen therapy, permanent pulmonary fibrosis is a major complication.

2. Its frequency is directly related to the large total doses delivered to the lung by divided dose technique and the area of lung tissue irradiated.

3. It is not apparent from this study that age is a predisposing factor.

4. Based on the studies of the alterations of pulmonary physiology in these patients, the dyspnea is due to: (*a*) marked diminution of the vital capacity caused by the fibrosis itself, (*b*) fixity of the chest wall, (*c*) compensatory pulmonary emphysema, and (*d*) hyperirritable Hering-Breuer reflexes.

5. The dyspnea may be relieved by means of an abdominal belt or weight reduction or both.

6. The cough improves as the dyspnea decreases.*

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* For discussion see page 758.

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THE INADEQUACY OF SIMPLE MASTECTOMY IN OPERABLE CANCER OF THE BREAST*

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BLOODGOOD[5], in 1935, stated that the type of surgery for malignant tumors of the breast was a settled question. Dean Lewis,[21] in the same year, believed that a tendency was developing toward less radical surgery for treatment of cancer of the breast. Since then, publications in favor of simple mastectomy and others which question the routine use of radical mastectomy have appeared in journals of this country and abroad.

If an essay bearing a title such as the one that heads this presentation had been read shortly after the turn of the century, during the active days of Halsted, it would have been regarded as something written in jest, deliberately to provide amusement. Today this paper is not the superfluous contribution it would have been several decades ago for there are those, surgeons and radiologists alike, who believe that simple mastectomy is the operation of choice in early operable cancer of the breast.

The accomplishments in the surgical treatment of mammary cancer are not as meager as some believe. Starting from a period in the past century where almost no one could report a cure in an established case of cancer of the breast, progress has been gradual and constant.

In 1801, Joseph Adams,[4] after years of experience, concluded that even the early and mild cases of cancer of the breast were incurable. In those days surgery varied in extent from local excision to simple mastectomy.

In 1815, John Rodman[32] was led into writing a monograph concerning cancer of the breast in an effort to learn "how there possibly could be so many contradictory opinions entertained by men of high character, in fact, by the most respectable members of the profession." A study to explain present contemporary diametrically opposed opinion in the treatment of cancer of the breast could be a modern pretext for the compilation of a text.

In the days before Wells, Long, and Morton, anesthesia was not known. Surgical procedures, if attempted, were done in great haste. The criteria of operability, if we believe the writers of that time, were at least as stringent as they are today. Because of the numerous opponents to operation, surgical interference was considered perhaps in a narrower group of patients than today, yet Rodman remarked significantly that the lack of success in removing mammae for early tumors when the prospects for cure appeared excellent deterred many surgeons from performing the operation. The operation varied from simple excision of the tumor to complete removal of the breast.

The well known Paget[27] was asked to give his opinion on the curability of cancer of the breast by operative removal of the organ. He answered: "I will not say that such a thing is impossible but it is so highly improbable that a hope of a cure occurring in any single case cannot be reasonably entertained."

After the advent of anesthesia, surgical procedures in general were more frequently performed.

Virchow published his work on cellular pathology in 1858. Nine years later, Moore,[24] of London, pleaded for more radical surgery, and was probably the first to give us a conception of the pathology of cancer of the breast as we understand it today. Sepsis in the days of Moore probably did much to discourage more radical intervention in behalf of the patient. Of 167 hospital breast extirpations done by Velpeau[37] prior to 1854, the mortality rate was

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19 per cent, with sepsis the most frequent cause of death. As late as 1879, William Nunn[25] states that if no obvious involvement of the pectoral fascia is found, it should be left intact. He considered this an important means of limiting sepsis. Volkman[38] ignored sepsis and recommended the removal of the pectoral fascia in every case. He made it a rule never to be misled into conservatism, even for the smallest tumors. These were pioneering days when men for the first time were thinking scientifically and attempting to improve technique on a basis of knowledge. Yet, in these very days of improvement, Gross[11] did not save one patient in his first hundred cases. Sands[34] told Halsted he could not boast of having cured a single case. They were doing simple mastectomies.

Following the development of antiseptic surgery, later displaced by aseptic surgical technique, a marked reduction in the incidence of sepsis and decreasing surgical mortality rates were soon noted. The operation of radical mastectomy, now made a technically safe procedure, was conceived and presented almost simultaneously in 1894 by Halsted and Willy Meyer. Radicalism in mastectomy ran wild; in some cases the entire lateral half of the shoulder girdle and upper extremity were sacrificed. Two problems arose. First, it became necessary to establish just what cases were to be considered operable, or, in other words, at what stage in the extension of the disease the surgeon should refrain from attempts at curative surgery. This could be learned only by experience. The momentum of early enthusiasm swung the pendulum to the extreme of radicalism. The early surgeons soon learned that nothing was to be gained by operating upon patients where the disease had progressed beyond the axilla. Since then other contraindications have appeared, but, broadly speaking, the definition of operability has remained essentially unchanged down to the present day. The second problem was the need for definition of adequate surgery for cancer of the breast; in other words, just how far should a surgeon go beyond excision of the gross tumor, in the absence of clinically demonstrable disease. This question was soon answered by the adoption of the fundamental principles of the classical Halsted operation, modified later in detail by Handley and others. Unfortunately, the degree of radicalism routinely to be employed for all operable cases of cancer of the breast is not today the settled question that Bloodgood asserted it to be five years ago. Some surgeons, unmindful of the gains made in the treatment of cancer of the breast and unduly discouraged by the results of surgery or unduly encouraged by the results of irradiation, have fallen away from the standardized and generally accepted radical mastectomy.

What are some of the possible reasons why one performs a simple mastectomy in operable cases of cancer of the breast?

1. There are surgeons who believe that in early, local cancer of the breast simple removal of the breast itself eradicates the local disease; that more extensive surgery is unnecessary and irradiation useless.

2. There are those who believe that in early, local cancer of the breast simple mastectomy plus exploration of the axilla, followed by irradiation, is adequate treatment.

3. There are those who believe that cancer of the breast is curable only when it is still localized to the breast, and therefore they believe that simple mastectomy is adequate. If microscopic spread has taken place beyond the breast proper, they believe that radical surgery is of no avail.

4. There are those who believe that in cases of operable cancer of the breast, with palpable axillary nodes, simple mastectomy plus irradiation is adequate treatment.

5. There are those who believe that cancer of the breast is adequately treated by various degrees of conservatism in surgery, short of the complete operation.

It is beyond the scope of this paper to discuss singly and in detail the variations in the conservative, surgical treatment of operable cancer of the breast. We shall

therefore limit our remarks, first, to the evaluation of simple mastectomy in clinically early cancer of the breast, and, second, to the evaluation of simple mastectomy complemented with irradiation in cancer of the breast with palpable axillary nodes. We wish to show thereby that the radical operation is the only method of excisional surgery routinely to be employed in the treatment of operable cancer of the breast.

If a surgeon believes that simple mastectomy is adequate treatment for early, local cancer of the breast, his first task in the interest of his patient should be to show that, in a given selected case, he is dealing with an early cancer. This brings up the question: What is an early cancer? Here again we must be brief in answer. This question could be divided into two parts:

First, what is an early cancer of the breast pathologically? In our present stage of development, the consensus among pathologists is that an unequivocal diagnosis of cancer in a given microscopic section can be made only where actual invasion can be seen. It follows that these are not strictly early cancers, for surely at some time during its development a cancer need not show invasion. Some pathologists believe that a cytological diagnosis of cancer is possible, but at present this opinion is opposed by a formidable group of distinguished and experienced men. We may say, therefore, that cancer can be recognized microscopically only after the development of the characteristics which inspired its name many hundreds of years ago—not in reality early cancer at all.

Second, what is an early cancer of the breast clinically? Given a patient with a clinically local, freely movable tumor of the brest, can one state that the tumor is early because of its recent discovery by the patient or because of the small size of the tumor? The tumor recently discovered by the patient is new only so far as her knowledge of its existence is concerned, but actually it may have been present as a lump for months or in some cases even for years. The size of the tumor is not necessarily or irrefutably a measure of its recent development or of its local character. The rate of local growth and the speed of clinically indetectable microscopic spread in different tumors of the breast vary between wide limits. For example, a patient, shown at the Tumor Conference at the Philadelphia General Hospital recently, suffered from multiple osteoclastic lesions involving every bone in her skeletal framework. At postmortem these universally disseminated lesions were proved to be metastatic from a hazelnut-sized, movable tumor of the breast, of which the patient had been unaware. Many more similar examples could be quoted.

Further and more convincing evidence can be brought forth to show that one cannot safely make a clinical diagnosis of early cancer of the breast. Harrington[16] shows in his series that 29 per cent of the cases clinically thought to be free of axillary metastasis actually have proved axillary involvement. Lund,[22] of Boston, states his experience and places the figure at 40 per cent. Lane-Claypon,[20] in her careful studies, shows that of all cases clinically diagnosed as Group I cancer of the breast, that is, without clinical evidence of axillary cancer, over 50 per cent of the cases actually were Group II P, with pathologically proved metastasis to the axilla. Now, if between 29 and 50 per cent of all cases diagnosed as Group I actually are not Group I at all, but Group II, the difficulty, indeed the futility, of attempting to select early cases for simple mastectomy can be recognized.

Evidences from many sources, therefore, emphatically demonstrate that axillary cancer cannot be assumed to be absent, on the basis of a negative clinical examination.

In spite of the evidence cited above, Fitzwilliams,[9] English writer of a textbook, who a few years before commented on the difficulty of early diagnosis, makes this amazing statement in 1937: "The early diagnosis nowadays possible enables us to treat cases at a far earlier stage than ever before and makes partial removal of the breast just as safe as the partial removal of a limb or

tongue without the wide mutilation which is so important to a woman. In the first stage, it is only logical to treat a local disease by local operation. . . ." Consider this statement in the light of the statistics just quoted.

An equally forceful argument to show our inability to select early cases of cancer can be found in a comparison of the results achieved by simple mastectomy and radical mastectomy.

It is true, and can hardly be denied by anyone, that should every cancer cell in a given case of cancer of the breast be completely eradicated, the patient should be cured, regardless of the means employed to accomplish this end. In what percentage of cases does simple mastectomy fail to effect this desired eradication of the disease?

In a corrected series,[20] in which simple mastectomy was done for early, local cancer of the breast, 54.6 per cent were dead after five years. In another series (operable cases) of 40 simple mastectomies, followed by irradiation, 56 per cent were dead after five years.[10] The average five year survival rate for simple mastectomies varies from 38 to 50 per cent.

Even these palpably poor results cannot fairly and properly be submitted for statistical comparison with the end-results of radical surgery. One must bear in mind that the reported series are small and the cases highly selected. All series include many cases of slowly growing tumors, present for years in the breasts of old women. In addition, some of the patients were operated on for benign conditions of the breast, and a cancer was inadvertently discovered after careful study by the pathologist.[20] Not only were the cases highly selected but in some instances cancer was not even suspected clinically.

Simple mastectomies are performed because the surgeon believes he is dealing with local cancers which can be easily cured. Theoretically, if these were, in fact, local tumors, he should cure 100 per cent; yet the five year survival rate is only 38 to 50 per cent. The deduction here is that the error in clinical ability to diagnose early cancers is over 50 per cent. Over 50 per cent of cases thought to be early, and therefore supposedly curable by simple mastectomy, have spread beyond the conservative operative field. There are those who will answer this by saying that, if the spread of cancer has advanced beyond the simple mastectomy field, microscopic advance is probably also beyond the operative field of a radical mastectomy. The fact is, many cases of cancer which have spread beyond the operative field of a simple mastectomy will not have spread beyond the operative extent of a radical mastectomy. Contrast the best comparable results of simple mastectomy against the best end-results of radical mastectomy. For simple mastectomy in Group I C cases (highly selected), the best five year survival rates are about 38 to 50 per cent. For radical mastectomy in Group I P cases (taken consecutively without selection), the best five year net survival rates range from 81 to 93 per cent.[20,26] From these results one can interpret that 50 to 60 per cent of the cases thought to be early have spread beyond the operative field of simple mastectomy; that 30 to 40 per cent which have spread beyond the reach of the conservative operation have not progressed beyond the reach of the radical operation; and that only about 10 to 25 per cent have spread beyond the operative extent of a radical mastectomy. This should be more than answer enough for those who insist that the curability of cancer of the breast is governed only by its local character; that the extent of the operation is unimportant; that if simple mastectomy fails, the case is hopeless.

Further evidence can be submitted to show that we should plead for improvement in the operating technique of radical mastectomy rather than to argue the inadequacy of simple mastectomy.

The British Ministry of Health Report, Number 34, records the end-results of a select group of distinguished surgeons which show 92.3 and 90.1 per cent net five year and ten year survival rates in Group I

P cases.[6] These figures, which are considerably better than the average end-results for radical mastectomy, strongly suggest that in the hands of those most competent to perform a truly radical operation brilliant results can be obtained. It is reasonable to hope that with general improvement in technique, material improvement in end-results will follow.

A recent critical study of the end-results of radical mastectomy shows that, among comparably competent surgeons on one hospital staff of a large medical center, those having a longer average operative time show impressively better end-results than their more speedy colleagues who were also doing radical mastectomies.[31] The deduction here is that simple mastectomy is totally inadequate; that even a carelessly performed radical mastectomy considerably improves results; that a carefully performed complete operation gives by far the best results.

A few years ago, at the Oncologic Hospital, a few selected cases of cancer of the breast were managed by simple mastectomy. Some cases were given roentgen therapy in addition. Because of early recurrence and generally poor results, the practice was discontinued. We have a series totaling 29 cases. Of these 29 patients, 8, or 27.5 per cent, were alive an average of twenty months, without evidence of disease when last seen. Seven (24.1 per cent) were dead, having lived an average of twenty-two months postoperatively. Fourteen (48.3 per cent) developed recurrences six weeks to thirty-seven months after operation, averaging twenty-two months. In the past two years, 6 patients have been seen at the Oncologic Hospital with local recurrence following simple mastectomy performed elsewhere. They were treated by secondary radical operation and roentgen therapy. The end-results in such cases are known to be poor, as the patient has already lost her best chance.

If one believes he can improve his results in simple mastectomy by exploring the axilla, he again is mistaken. In the first place, the axilla cannot be properly cleaned without severing the muscles. In the second place, important lymphatic routes are present in the pectoral fascia and pectoral muscles. Last and most important, Lane-Claypon[20] shows the end-results in these cases to be even less impressive than the end-results of simple mastectomy alone. A patient with the axilla left undisturbed is better off than she would be after a simple exploration of the axilla or an incomplete dissection.

No one denies that a truly early, local cancer is curable by simple mastectomy, or by even more conservative measures, if the entire disease is removed. We have shown, however, that one cannot, with any degree of accuracy, select early cases of cancer of the breast for management by conservative operations. It appears that surgeons who perform simple mastectomy in the belief that an early cancer can be entirely eradicated have worked from a false premise.

Clearly, one cannot feel justified in attempting to cure cancer of the breast by simple mastectomy in the belief that he is operating on an early cancer of the breast. The proved, present inability to determine accurately the absence of axillary metastasis and microscopic spread elsewhere within the limits of radical surgery denies anyone the right lightly to choose any but a proved mode of treatment in so-called early cancer of the breast. To ignore this dictum means the sacrifice of 30 to 40 per cent or more of such patients operated.

Let us now consider carcinoma of the breast with palpable axillary nodes (Stienthal II) and its treatment by simple mastectomy and irradiation.

One who believes that simple mastectomy, plus irradiation, is adequate treatment must believe, first, that his operation does not completely remove the cancer; second, that more extensive surgery is useless, and that irradiation is a more dependable instrument than the scalpel for destroying the remaining cancer.

We have already shown that in a high percentage of cases metastasis to the axilla has taken place, even though clinically un-

demonstrable. Let us stress another important point concerning axillary nodes. Given 100 operable cases of cancer of the breast, with palpable axillary nodes clinically diagnosed as metastatic, it has been shown that from 10 to 32 of the cases will not have cancer in the axilla.[15,22] The nodes are purely hyperplastic or inflammatory. From 10 to 32 per cent of Steinthal II cases are therefore actually Group I P, or pathologically proved Group I cases. But the absence of microscopic evidence of cancer in the axillary glands in a given case does not permit one to conclude that he is dealing with a local tumor of the breast. The true picture of cancer of the breast is a tumor in the breast proper from which a centrifugal microscopic spread takes place. The extent of this spread cannot be determined clinically, and can never be assumed to fall within the operative limits of a simple mastectomy, even though the axilla is shown to be uninvolved. This has been shown earlier in this paper by the failure of simple mastectomy to eradicate the disease in a high percentage of cases. For cases falling in this category radical mastectomy is curative and decisive, while simple mastectomy with postoperative irradiation is palliative and extenuative, as will be shown later.

Now we are to concern ourselves with cases that actually do have metastasis to the axilla. Is simple mastectomy, followed by irradiation, the method of choice for this group? At this point it becomes necessary to determine the ability of irradiation to perform its given task, namely, complete destruction of all cancer cells within the compass of radical mastectomy. The evaluation of irradiation employed in such cases must be based on its proved ability completely to destroy cancer. Here we wish to clarify our position. The value of roentgen rays or irradiation in general as an adjuvant in the treatment of operable carcinoma of the breast is firmly established. It should be emphasized that postoperative irradiation is used routinely by us. We do not feel as some do that, if irradiation is to be of value at all, it should be able to destroy cancer entirely without the aid of surgery. We hold ordinarily that the ability of irradiation completely to destroy cancer is not the only criterion of its value. It appears, however, that should one advance the proposition that simple mastectomy, plus irradiation, can achieve as satisfactory results as radical mastectomy, with or without irradiation, the proponent of this thesis must demonstrate that irradiation is always capable of completely destroying cancer cells in lymph nodes and elsewhere within the limits of the operative field. To put it differently, he must show that the roentgen rays can accomplish at least the equivalent of surgery, namely, the destruction of all the cancer cells that radical mastectomy can remove.

Let us examine the question briefly. In general, it is agreed that many, if not most, mammary cancers are about as radioresistant as epidermoid carcinomas.[8] If roentgen treatment is delivered to a wide area of the thorax and axilla in doses known to be insufficient to result in total destruction of a superficial epithelioma which is closely comparable to most mammary cancers in its response to irradiation, does it not follow that such treatment is insufficiently reliable to be depended upon as the sole agent of treatment?

Schinz[35] in 1940 stated that the number of cures from irradiation was small in his experience, but that we should not underestimate the palliation accomplished.

Howes and Bolker,[18] in 1940, reported their experience with the use of intensive deep roentgen irradiation (2,800 r to each of two fields, crossfiring on the breast, and occasionally a third field of 1,400 r—200 r daily to each of two fields). They were able to produce pronounced effects on the breast tumor, but mention only 2 cases in which the cancer was entirely destroyed. In their experience, changes in neoplastic axillary nodes that could be attributed to irradiation were slight, despite the unusually intense axillary dose averaging 2,800 r to each portal. These authors point out significantly that radiation treat-

ment of cancer is still feeling its way.

Even more convincing evidence is available to show that external irradiation alone cannot accomplish the equivalent of surgery. In a series of 47 cases treated at the Oncologic Hospital with moderate to intensive irradiation, our pathologist, Dr. J. McFarland, showed that in 16.2 per cent of the cases all the cancer cells in the sections studied were destroyed; in 35.2 per cent the cells were moderately affected; in 37.8 per cent the cells were markedly affected, and in 10.8 per cent the cells showed no changes attributable to irradiation. Many of the cases showed active mitosis, even in the presence of marked irradiation changes.

Cheatle and Cutler[7] state that attempts to treat mammary cancer by using maximum external irradiation demonstrated marked degenerative changes, but complete sterilization was not accomplished. They conclude that complete devitalization by external irradiation in most cases is probably impossible.

Adair,[3] in 1935, believed he had destroyed breast cancer by use of the radium element pack in only 28 per cent of 39 primary operable cases. In a similar group of 42 operable cases treated by deep roentgen therapy, the breast cancer was destroyed in only 16.5 per cent. He cites the inability to destroy axillary nodes in a very high proportion of cases. At that time Adair was said to have given up all attempts to treat this disease by external irradiation alone, because of failures.[2,3] More recently, Adair[1] reports up to 50 per cent tumor destruction following intensive external irradiation, and only 22 per cent sterilization of the axillary contents. These are immediate results. The presence of previous axillary metastasis in Adair's series was determined by typical scarring, which he and others believe to indicate previous cancerous involvement. McFarland[23] does not accept this as irrefutable evidence of previous cancerous disease. It is possible that the figure 22 per cent is high.

Some may gain the impression that we believe that a dissection of the axilla is always capable of sterilizing the region of tumor tissue. This is not our position. We do believe that, given the choice of two modalities—one which is effective in only 22 per cent of cases, the other which obviously destroys all cancer that can be surgically removed—it is logical to deduce that a combination of the two modalities is safer than sole reliance on a 22 per cent chance.

Simple mastectomy followed by external irradiation cannot replace radical mastectomy in Steinthal 11 cases. Can simple mastectomy followed by interstitial irradiation used alone or jointly with roentgen irradiation take the place of radical mastectomy? Before answering this question, the following is parenthetically inserted.

Only if the end-results in proved cases are comparable or better than the end-results of radical mastectomy can interstitial irradiation be recommended as a sole method of treatment for cancer of the breast with palpable axillary nodes, or as a complementary treatment to the lymphatic drainage area following simple mastectomy. Interstitial irradiation cannot be offered to a patient as a non-surgical measure. It is not a substitute for surgery. It is a surgical procedure which requires special training, and is hardly less trying to the patient than radical mastectomy. Convalescence is longer; scarring and retraction of the tissues sometimes produce a less desirable cosmetic result than radical mastectomy. The complications vary. Neuritis, sepsis with elephantiasis or limitation of arm motion, rib necrosis, and death are reported.

In attempting to treat with interstitial irradiation a large area such as is required in cancer of the breast, one of the fundamental requisites of radiation therapy cannot be met. An equable radiation dosage which is everywhere carcinocidal is certainly a theoretically difficult, and, from a practical standpoint, impossible feat. Various techniques have been employed in an effort to obtain uniform interstitial irradiation. None of the methods has been practical, and significant is the fact that most

therapists employing interstitial irradiation as a routine procedure have abandoned it.

Keynes has probably had more experience with interstitial irradiation than any other of the few men employing this method. He recommends the surgical removal of the primary tumor, followed by interstitial irradiation of the drainage area, including the axilla. The primary tumor which he can feel and accurately needle, he removes; the drainage area about which he can have but meager knowledge, he attempts to treat. Singularly, Keynes admits that his needles are frequently improperly placed.

The ability of interstitial irradiation to destroy cancer cells everywhere within the operative field has not been demonstrated. Almost no experimental work in this direction has been done, and it is an obviously difficult problem. Teahan[36] recently reported 7 cases treated by simple mastectomy, followed by interstitial irradiation of the axilla. In only one case was the axillary disease controlled.

It is well known that the primary tumor is usually more radiosensitive than the metastatic foci, yet in 1939 Ross,[33] following the technique of Keynes, reported a residual palpable tumor of the breast in 37 of 50 cases treated by interstitial irradiation. Significant is the fact that those using this technique almost routinely excise the breast tumor.

More striking is the following statement made by Regaud:[30] "I have completely given up the use of interstitial radium for primary operable breast cancers because of poor results and danger of spreading the disease."

Keynes[19] is able to show 65 per cent and 51 per cent five and seven year crude survival rates in Group I cases, and 26 per cent and 18 per cent five and seven year crude survival rates in Group II cases. These are the best end-results reported. An obvious sacrifice of life can be noted even without the application of correction factors for the many unbiopsied cases.

Lane-Claypon[20] and others have shown that a five year period is inadequate to determine the end-results of radiation therapy because growth restraint alone will place many patients in the five year group who will later succumb to the disease. The mortality rate in the second five year period far exceeds that of radical mastectomy, indicating that irradiation brings about growth restraint but fails to destroy the cancer in a high proportion of cases. This must be considered when the end-results of irradiation and surgery are compared. It may be added that the difficulty of evaluation of the nodular scars in the irradiated area may place many patients in the five year group who undoubtedly have cancer unrecognized because of retraction, scarring and nodularity resulting from the interstitial irradiation.

Few investigators have had actual large experience with the interstitial treatment of cancer of the breast. Probably the total reported number of patients so treated is 500 or less. None of the few original authors has advocated that this form of treatment should displace the radical operation. Some enthusiastic readers of the original articles, with no personal experience or with little appreciation of the difficulties involved, are those who unconditionally endorse this form of treatment and publish recommendations advocating the abandonment of radical surgery.

Interstitial irradiation does not appear to be able to perform the equivalent of surgery, namely, the destruction of all the cancer tissue the radical operation can remove. The adjuvant use of external irradiation with interstitial irradiation can add but little because of the known inability to supply sufficient external irradiation to the axilla. Theoretically, some areas may be so under-irradiated that the additional external irradiation may not supply the necessary carcinocidal dose. We wish to state again that we still refer to the field where the existing cancer cells could be completely removed surgically. From reports thus far published, from 50 to 75 per cent of breasts examined after radical irradiation for

known cancer still contain viable cancer cells.[26] Adair[2] reports 36.3 net five year survival rates in a selected series of 37 cases.

The high percentage of five year survival rates attained by irradiation reported occasionally in the literature is in direct disagreement with the foregoing discussion.[10,29,39] There must be some explanation for this divergence of authoritative opinion. Perhaps most important is the question of diagnosis. Because of extreme case selection and the high percentage of error in clinical diagnosis, radiologists today admit that the end-results of unproved and perhaps improperly classified cases treated by irradiation cannot be compared with the end-results of proved cases treated surgically. The end-results achieved by irradiation in the treatment of clinically diagnosed cancer of the breast have impressed no one, because of the known high percentage of error in diagnosis. Halsted,[13] in the days when few local cancers were seen, had an error of 10 per cent in diagnosing tumors as malignant, even with the surgical specimen in his hand. Haagenson[12] states that only 50 per cent of all lesions of the breast are accurately diagnosed by experienced men. At the Mayo Clinic, in a study of 100 breast cases where early cancer was considered as a possibility, 73 per cent were found to be benign. Adair lists eleven different types of lesions that may simulate cancer of the breast. Many radiologists today agree that biopsies are necessary before their statistics can be compared.

There is not space here to discuss further the various factors which should be considered in evaluating the end-results achieved by irradiation. Suffice it to say that no reasonable comparison can be made until the rigid standards adhered to in reporting surgical end-results are adopted.

It appears likely that the benefits obtained today from roentgen therapy in cancer of the breast are not so much from destruction of cancer cells as from incarceration of residual cells in fibrous tissue, with resultant growth restraint. One concludes that irradiation cannot be substituted for the scalpel; that simple mastectomy complemented by irradiation in any combination cannot take the place of radical mastectomy followed by irradiation.

It may be said that more than four decades after the discovery of the roentgen rays and radium their place in the treatment of cancer of the breast has not yet been settled. Irradiation of cancer of the cervix uteri and corpus uteri has become standard treatment, and is so accepted by radiologists, general surgeons and gynecologists. The general discontinuance of operative removal of cancers of the uterus eloquently indicates the willingness of the surgeon to adopt whatever method is shown to be for the best interest of the patient. If and when the end-results in the treatment of operable cancer of the breast by irradiation or by simple mastectomy and irradiation equal or surpass the results achieved by the radical operation, with postoperative irradiation, we believe the surgeon will recognize the fact and act accordingly.

In the present stage of development it is yet premature for anyone to recommend that irradiation in any form has replacement value for surgery. Radiologists are keenly aware of the discredit that may befall them if overenthusiasm is permitted to endanger the acknowledged progress they have made. We regard this as a healthy attitude to adopt during this search for better and uniform technique. We wish to emphasize again that our efforts to show that radiation therapy cannot replace the radical operation should not be construed as an indication that we doubt the worth of radiation therapy as a valuable adjuvant in operable cases, and as the primary treatment of choice for inoperable cases. Where surgical treatment is refused or if absolute contraindications exist, naturally irradiation or conservative surgery or both should be employed.

Here we recall the admonition of Pfahler:[28] "The radiologist must be trained as and assume the responsibility of a consultant. Some . . . patients will consult the

radiologist rather than the surgeon. We must not let this fear of operation influence our judgment and choose to treat a patient (by irradiation) who can be better treated by surgery." Only those men with a broad and unbiased perspective can give the patient her best chance for life.

The American Society for the Control of Cancer, its Women's Auxiliary, the medical profession in general, interested social workers and laymen are trying to teach women that early cancer is curable; that there should be no delay in seeking the advice of a physician following the discovery of a lump or any sign or symptom that may indicate the presence of a cancer.

The educational program is bearing fruit as can be shown by the increasing percentage of patients visiting physicians before axillary metastasis is demonstrable. Halsted in a series of 133 patients could show only 6 in which axillary metastasis had not occurred.[14] Today the Free Harvard Commission shows 51 per cent without axillary metastasis.[17] Harrington[16] mentions that, in 1916, 74 per cent of his cases had axillary metastasis, whereas in recent years there has been a progressive decrease in the percentage of patients seen with axillary metastasis at the Mayo Clinic. In 1938, 51.1 per cent of Harrington's cases did not have axillary metastasis. There are other reports which show over 40 per cent of patients without axillary involvement. These are encouraging signs, in spite of what some pessimists may believe. There is no reason why these figures should not become universal, and, for that matter, they should improve.

The management of Group I P cases is an important problem comprising from 30 to 50 per cent of the total cases reported in many recent series. We may rest assured that we are going to see cases of cancer of the breast earlier and earlier after their discovery. We are therefore going to see more and more tumors with less and less evidence that they are malignant. We must not be lulled into conservatism by the diagnosis of so-called early cancer of the breast. The lay public is being convinced that early cancer of the breast is curable, but that claim has been based on the results obtained by master surgeons doing radical mastectomies (without postoperative irradiation), not on the results of conservative surgery.

We are all aware of the recent, progressive increase in the mortality rate of mammary cancer. It is true that increase of the female population over fifty years of age has some bearing on the increased incidence and mortality rate. This fact cannot, however, account entirely for this increased mortality. It has been properly suggested that the increasing mortality rate of mammary cancer could be due in part to the increasing lack of certainty in the minds of some as to the proper therapeutic management of cancer of the breast.

We advocate the radical operation in all Stage I and Stage II cancers of the breast, and shall continue to use it until conservative procedures are proved equally efficacious. We do not recommend the continued advocation of methods, the merits of which have not yet been established.

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DISCUSSION OF PAPERS BY DRS. LEACH, ET AL., AND BRANSFIELD AND CASTIGLIANO

DR. SOPHIE SPITZ, New York. Dr. McIntosh and I have been interested in the subject of fibrosis of the lung since 1933. In all that time we have certainly not evolved anything as practical, useful and well reasoned in the way of treatment as Dr. Leach has presented, nor indeed has anyone else. The simplicity and availability of the abdominal belt commend it as much as does the sound physical rationale established for its use.

We would be interested to know whether the belt is as successful in fibrosis arising from heavy irradiation of the base of the lung or mediastinum. It seems to be established that there is a greater morbidity from heavy irradiation of the mesial lung and mediastinum than from the usual types of breast treatment where the bulk of the cross-firing falls more peripherally.

Dr. Leach states that "we have been unable to determine any significant correlation between the symptoms, signs, degree of fibrosis and amount of radiation to the lung."

Our conclusions on these points are somewhat different and it might be of interest to re-state them due to the fact that our technique of treating lesions of the breast and our changes of technique ran almost parallel with the work at Memorial Hospital and our method of computing lung tissue dose was the same.

Our reported series of cases was smaller—

only 60—but chest films were made immediately before and after treatment every month for the first six months, every three to four months for the next six months, and twice a year thereafter as long as followed. This allowed of more accurate deduction as to correlation between lung tissue dose and degree of fibrosis as shown on the film, than if films were made only in the presence of symptoms.

In our 60 cases, 36 showed definite lung changes and only 11 showed symptoms. On a symptom basis, the other 25 would not have been included or any attempt at deduction made from them. And these 25 were not all mild reactions. One absolutely symptomless individual had the highest lung tissue dose in the series—9 threshold erythema doses at one point.

I do agree heartily that the correlation between symptoms, on the one hand, and depth dose and film changes, on the other, is most insecure, but believe the correlation much more dependable as between depth dose and film changes with modification for age.

Here, too, our studies differ, showing a definite relation between age and permanent lung changes. We had a mean age similar to that stated by Dr. Leach—forty-six—and we had a greater number of pneumonitis cases in this intermediate age group but a proportionally larger number of the Grade III and IV reactions in the older cases even though not always with more severe symptoms.

However, while I cannot forego stating my convictions as to what I believe are rather steady correlations among the factors of lung tissue dose, lung changes as registered by films, and age, I consider that Dr. Leach and his associates have taken a long stride ahead of all the previous studies on the subject with their original and valuable physiological and therapeutic conclusions.

Dr. George M. Dorrance, Philadelphia. I am glad to discuss this paper on the "Inadequacy of Simple Mastectomy in Operable Cancer of the Breast" because several of the cases which led the authors to reach their very definite conclusions on the inadequacy of simple mastectomy in early cancer of the breast resulted from the work done at the American Oncologic Hospital when I turned over the breast service to Dr. Bransfield three or four years ago.

When I first started the breast clinic at the American Oncologic Hospital twelve years ago, I insisted upon radical mastectomy on our early cases but a number of the cases left us—some insisted upon simple mastectomy to be followed by irradiation while others only wanted roentgen or radium treatment. These procedures were quite common in many of the clinics in Philadelphia at that time. Because of this fact, against my better judgment in some selected cases, I performed a simple mastectomy rather than have the case leave the clinic; other cases were treated with roentgen rays or radium, and in some cases simple amputation was done; a number of cases were treated exclusively by roentgen rays and radium, or subsequently treated with a simple mastectomy did remarkably well and appeared to be free of the disease for varying periods—three to seven years.

Local recurrences, however, did result, and about this time Dr. Bransfield took over the service and he decided to do a radical mastectomy on every case he considered operable, regardless of the previous treatment. In every case, the pathologist was able to demonstrate malignancy. In 4 cases, there was no clinical evidence of it. Last year, in our clinic, we had 6 cases of simple mastectomy done in other clinics all of whom showed local recurrence or involvement in the axilla. A radical mastectomy was performed in all of these cases.

I am convinced from the work in our clinic now that simple mastectomy should never be done in early cases. These cases are entitled to radical surgery, and the sooner it is done the better. I am speaking of early cases and not simply where simple mastectomy is done as a palliative measure. I agree with the authors that it is impossible to diagnose cancer of the axilla clinically and feel there is an error of 50 per cent in the best clinics.

I shall not discuss preoperative and postoperative irradiation. I am not exactly sure where I stand on preoperative irradiation, but I have used it a number of times in selected cases. However, I do use postoperative irradiation in all cases.

Now, in regard to fibrosis, I feel, as all of us do, that we have just heard a remarkably good presentation of this subject and there is little I can add. We have been doing some experimental work on rabbits at the American Oncologic Hospital trying to produce fibrosis of the lungs. We are not ready to report on it. As a matter of fact, we have had such contradictory

results that we may have to repeat the whole experiment before making a report. From the purely scientific standpoint, it is too bad that we do not see fibrosis in the breast cases which come to autopsy. The majority of these cases get well of their fibrosis and hence we lack the benefit of the pathological reports. One thing is certain, one cannot predict with any degree of accuracy what type of patient or what age of patient will develop post-irradiation fibrosis. The amount and type of roentgen treatment given, while it must be a factor is certainly not the only factor in causing this condition.

DR. FREDERICK W. O'BRIEN, Boston. I would like to tell Drs. Leach and Bransfield how much I have enjoyed their papers. If we have time, I would be interested in having Dr. Leach give us a description of his grades of fibrosis and also what the roentgenograms show in those grades.

I was particularly interested in Dr. Bransfield's paper. At the outset I would like to say that other things being equal, I also believe that radical mastectomy is the proper procedure in cancer of the breast, but would like to call your attention to the fact that when surgeons report cases, they are usually reporting survival rates on selected cases. It would be fine if they would adopt a standardized terminology. One talks of survival rate, meaning alive at the end of five years, another about cure rate and means without evidence of disease at the end of five years.

The radiation therapist doesn't often have a chance to select his cases, but the relative merits of surgery and irradiation alone or combined could be adjudicated rather promptly if surgeons and radiation therapists abandoned the Steinthal classification which is purely clinical and adopted something such as Portmann's which is a combination of the clinical and pathological.

The surgeon reports a 70 per cent cure rate in clinical I pathological I cases, i.e. those with no metastasis to the axillary nodes. Once that occurs the cure rate sharply drops to 30 per cent. This raises the question of the advisability of surgery in clin. II path. II cases.

The argument against irradiation in the summary of Dr. Bransfield's paper annoys me a little because I can't see how the radiation therapist can ever demonstrate what irradiation can do until he has had the opportunity. It is in the clin. I path. I group, the so-called favorable cases, that the surgeon shows a 30 per cent failure, chiefly due to the character of the tumor encountered and its method of spread.

These rapidly growing tumors with no demonstrable axillary nodes occurring most frequently in young women are radiosensitive and in my opinion should be treated preoperatively in an attempt to mobilize the tumor and improve the cure rate for this group.

Dr. Adair is here to correct me if I am mistaken. His work is constantly quoted to the effect that the fibrosis and skin destruction following irradiation is so grave that operation must be deferred from three to six months. My recollection is that when he started to do that work he had a definite point in mind, viz., to see how many carcinomatous breasts and axillary nodes he could sterilize.

I believe you can give preoperative irradiation which will benefit the patient and allow the surgeon to operate with impunity within two weeks, as Melnick and his associates have done. It is not necessary or desirable to attempt to give a cancercidal dose preoperatively.

DR. JOHN E. WIRTH, Baltimore. We are constantly being told that we should analyze our failures in order to find out what to do in the future to better our results.

I wonder if some of the discussants today could not profit by that remark as did the essayist and perhaps analyze their failures a little more to tell us how we should irradiate breast cancer, postoperatively or preoperatively.

We are told that we should give postoperative irradiation in that really small number of Group I cases. To what are the failures due? Are they axillary recurrences, supraclavicular recurrences, or due to distant metastases? I believe that in the majority of instances you will find that they are due to distant metastases, a small group due to supraclavicular metastases, and a smaller group to local recurrences.

To tell everyone to give postoperative irradiation doesn't make very much sense since one clinic will have a high percentage of local or axillary recurrences because they don't do proper radical dissections; another group who do excellent radical dissections will have very few local and axillary recurrences but death will be due to supraclavicular or distant metastases.

So I also think it is well to analyze the failures and if the individual clinic is getting a large percentage of axillary recurrences, then the axilla should be irradiated. If a certain percentage of them are supraclavicular, then also ir-

radiate the supraclavicular region, but if they are due mostly to distant metastases, I have yet to hear the answer.

Dr. Charles L. Martin, Dallas, Texas. The large number of cases with pulmonary fibrosis observed at the Memorial Hospital is somewhat of a surprise to me. In our own clinic we seldom see this complication in breast cases. This is probably due to the fact that we no longer strive to cure this form of cancer with roentgen irradiation, but merely attempt to augment surgical procedures by giving the largest safe doses to the tissue of the chest wall and axilla. Since the axillary nodes cannot be sterilized even when cross-fired through three portals we use only one portal directed into the axilla with the arm elevated. The chest wall receives tangential irradiation and another portal is directed at the base of the neck. When the method used is not likely to produce a cure it seems to me unwise to adopt a method which frequently produces roentgen fibrosis with its annoying symptoms.

Dr. Leach (closing). I wish to thank the discussers for their reception of this paper.

In answer to the question of Dr. Spitz concerning the correlation of the signs and symptoms, and the amount of radiation received by the lung, we believe that the symptoms depend not only upon the fibrosis evident in the roentgengram but, more important, upon the reduction of the vital capacity. This is the thing in the last analysis that provokes dyspnea. As the vital capacity drops, the increased ventilation resulting from exercise approaches the maximum possible ventilation for the given patient. In a patient with a severe degree of fibrosis, if the vital capacity is not reduced very much, that patient will not be dyspneic with effort.

Another important point to consider is the patient's psyche. Many patients are chronically short of breath with exercise, but the increased respiratory effort does not affect them to the point of complaining about it. Weight gain occurring after irradiation is an extremely important feature in the production of dyspnea. This occurs because their intake of food remains the same or increases although their activity decreases because of some exertion dyspnea. As the gain in weight grows larger the dyspnea increases.

We have been unable to correlate the physical signs with the degree of fibrosis in the roentgenogram. One is constantly misled, mistaking relatively little actual pulmonary change for advanced fibrosis. This is probably due to the diminished thickness of the chest wall and better conductivity of the denser tissues.

Concerning the predilection of the older patients to develop fibrosis more easily than those in the younger age group, we have not found this to be true in our group of patients. From a study of the roentgenograms of many patients, both young and old, we could not find any significant age difference.

Unfortunately we do not have adequate material for pathological study. Very few patients on the breast service with terminal cancer die in the hospital. Many of the autopsied cases have had complicating pulmonary metastases which immediately precludes use for study of radiation pleuropulmonitis.

In answer to the question concerning greater morbidity of pulmonary fibrosis, the nearer the process approaches the hilum of the lung; this is absolutely true since the threshold of irritability decreases from the periphery of the lung toward the hilum.

We have had opportunity to study a patient who was treated by the old massive dose technique in 1932. She had two cycles of 850 r separated by three months. She now complains of fever, constant cough and hemoptysis and progressive weight loss. She has a chronic diffuse pneumonitis in the affected lung. Bronchoscopy shows many areas of bronchial ulceration and other later radiation changes in the mucous membrane of the bronchi.

Dr. O'Brien asked our method of grading radiation fibrosis. Although we are somewhat unorthodox we grade them on the following basis according to the roentgenogram:

Grade I—Appreciable diffuse haziness of the lung field within the area treated.

Grade II—Heavy haziness and patchy infiltration. The mediastinum and/or the diaphragmatic leaf are drawn toward that area.

Grade III—Dense opaque pulmonary tissue within the treated area, and marked retraction of the mediastinum and diaphragmatic leaf toward the affected area.

Dr. Lenz asked the question concerning right heart failure in these patients. Except one patient in a study of this group and other patients there has not been one instance of right heart failure occurring in patients suffering from pulmonary fibrosis due to irradiation.

It is physiologically unsound to expect right heart failure in radiation pulmonary fibrosis unless one of three things has happened. There is either (1) such distortion of the mediastinum with angulation of the pulmonary artery or (2) mediastinal fibrosis or (3) metastatic disease is constricting the pulmonary aorta or obstructing the main pulmonary veins with resultant right ventricular strain and later failure.

Some time ago Wiggers pointed out that at least 60 per cent of the lumen of the pulmonary artery must be obstructed before right heart strain is evident. Considering one whole lung fibrotic, which rarely occurs, this would represent 50 per cent of its entire capillary bed, and this in turn is not equivalent to the resistance of occluding 60 per cent of the lumen of the pulmonary artery.

One cannot depend on electrocardiography to support statements of the frequency of chronic cor pulmonale since such changes are not due to cardiac changes but to changes of the position of the heart in relation to other intrathoracic structures.

DR. CASTIGLIANO (closing). I appreciate the opportunity of being permitted to make a few remarks. I think the important thing that all men interested in the problems of oncology should bear in mind when they make public statements, whether they be printed or heard by lay groups or organizations that will make it available to the lay public, is seeing that the remarks are made in such a way that there is no chance of being misunderstood.

In regard to diagnosis and simple mastectomy, in the hands of the most experienced men, error in diagnosis of cancer of the breast and the extent of the cancer is so large that I believe we should probably be a little bit less scientific and more empirical in the method of procedure. In other words, one should not apply his reasoning powers to a given case and say, "Now this should be treated in such a fashion." This has been pointed out by others; it is not original with us at all. I think if we treat problems in a general way rather than trying to determine what each patient should have, we may improve our end-results and certainly will be better able to interpret our results. In other words, if the leaders in the field can make errors of 50 per cent in diagnosis of breast conditions and up to 50 per cent in Group I and II cases, it is obvious that the average surgeon will probably make far greater average errors and be misled into simple mastectomy.

More than a few surgeons are performing simple mastectomies who are not talking about it. While traveling around the country, I have seen good surgeons doing simple mastectomies and saying, "Well I know I am not supposed to do this, but I think I am getting just as good results,"—impressions which I think have been disproved since the days of Halsted.

In answer to Dr. Lenz's question of what cases to operate upon, I don't believe that anybody follows any hard and fast rules of operability. We strive to do that, but we hold pretty much the position of Harrington that if we think we can benefit the patient by operation, we attempt a radical operation.

We agree entirely with Dr. Lenz in the statements that he made. I don't think there is any question in the minds of many here that even in cases where you do have metastases, that group itself can be broken down and the end-results for the two groups show a wide variation.

We have studied our end-results according to low lying axillary metastasis and axillary metastasis high in the apex and the presence of several large glands, and there is a difference. I think Bloodgood reported over 40 per cent five year survivals without evidence of disease in the low lying metastatic group, which certainly is a far more impressive figure than the 15 to 23 or 25 per cent for the general group.

I think that that is probably an argument in favor of surgery, because most surgeons ignore the effect on their statistics by accepting cases which they might otherwise have declined to operate on.

In regard to irradiation, we have no argument with anyone who wishes to treat his case with postoperative irradiation. We use that routinely and we firmly believe that it is of value.

In regard to what kills the patient with cancer of the breast, I don't know, but that could be discussed at great length. I think one thing that is very significant is the fact, as pointed out in the paper, that men who, by reputation and by fact, do perform the classical operation are able to show impressively better end-results,—I am talking about patients that live without evidence of disease for longer periods of time. This indicates that perhaps there must be something to the radical operation and to the regional metastases in relation to survival.

In an unpublished paper from a large clinic in New York City, where cases have been taken

alternately, the end-results show that the surgeons whose time has been longer at the operation have impressively better end-results, and it seems to me that if they were all doing radical mastectomies, certainly such fundamental things as the amount of skin removed, subcutaneous cleavage of the fascia, removal of the pectoralis major and pectoralis minor muscles, would be considered important.

All the surgeons in that hospital were probably employing a fairly uniform technique so that part of the operation was more or less classical. Where, then, was the additional time spent? I don't know but it seems to me that it might well have been spent in the axilla and those men who did spend additional time in the axilla were able to show definitely higher survival rates than the others. That is impressive and only a hint; it may or may not mean much.

In regard to the cases treated by irradiation and followed at a late date by secondary radical mastectomy, some of those patients went as long as five years before being subjected to a secondary operation. Now we know that secondary operation is associated with poor results in cases that have previously had simple mastectomy, followed by radical irradiation.

I am not so sure anybody knows just exactly what the end-results will be in cases that have been treated by irradiation, perhaps one or two or more cycles, over a period of three to four years. Many of these patients were questionable. We were not sure whether they had cancer or not. We felt they did have, and we gave them the benefit—or what we considered the benefit, at least—of a radical operation and we of course will not know for some time what the results in these cases are to be.

I don't believe there is such a thing as an early case of cancer of the breast, because certainly we can't demonstrate it pathologically. There are many pathologists today who insist that a diagnosis of cancer cannot be made in the absence of invasion microscopically.

Dr. MacCarty of the Mayo Clinic, and others, believe that a cytologic diagnosis can be made but an unequivocal diagnosis of cancer of the breast cannot be made without invasion. We have had that demonstrated time and again in published reports. So if we cannot make a diagnosis of cancer of the breast pathologically in an early case, how can we hope to make it clinically?

As to the borderline advanced cases, it is a matter of opinion as to how they should be treated and I think if one wishes to go ahead according to his judgment and use irradiation or surgery, it should be done.

ROENTGEN THERAPY IN OTITIS MEDIA*

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IN 1939 we published in this JOURNAL[1] the results which we had obtained by the use of roentgen therapy in otitis media. In our summary of this article we expressed our satisfaction with this mode of treatment and we have not altered our opinion since then. In this article we wish to give the results of further work in this field of therapy.

A search of the literature reveals that only two additions have been made since ours. Woodward[10] reports the use of roentgen therapy in 34 cases of acute otitis media which were given "adequate therapy," with no evidence of any definite benefit, but the author says that he has seen cases benefited by its use in the reduction of lymphoid tissue in the nasopharynx and about the orifices of the eustachian tubes. He has also seen results when this form of treatment was employed for polyps and granulation tissue in chronic purulent otitis media. On the other hand, Dowdy, Heatly and Pierce[5] report that this method is of distinct value in relieving the acute symptoms and in shortening the course of the disease. They employed this form of treatment in 30 cases, using one or more doses of 100 roentgens, and compared the effects with 26 controls. They found that in the acute, purulent, uncomplicated cases, the duration of the condition was shortened by six days, while in the complicated cases it was shortened by sixteen days.

In one of the reviews[11] of our previous paper, the comment was made that the effect of the irradiation was due to a decrease in the lymphatic tissue in the eustachian tube and in the middle ear, which allowed better drainage. As one of us has pointed out,[7] the eustachian tube opens 2 to 3 millimeters above the floor of the tympanic cavity, and so complete drainage cannot take place. Again, roentgen therapy has been employed in other types of acute and chronic inflammatory conditions where drainage could not take place, and the results obtained could be explained only by the process of breaking down, liquefaction, and absorption of the cellular débris. The work of such men as Cushway and Maier,[2] Tyler,[8] Desjardins,[4,5] Wintz,[9] and Milani,[6] has demonstrated this.

The technique used in this series is the same as was previously employed. It consists of 85 kv. (peak), 5 ma., 16 inch target-skin distance, and 1 mm. aluminum filter. The dosage was 50 to 60 r in infants, and in young children and adults the dosage has varied from 60 to 100 r.

In this series we are reporting 52 additional cases, which brings our total to 110. From Table 1 it can be seen that in the cases of acute catarrhal otitis media our results have remained unchanged, with the exception that the average dose has been increased. Several cases in this group have received this form of therapy more than once. Two patients have received four separate treatments and 2 patients three separate treatments. All of these responded readily without any complication, and as far as it can be determined, there has been no adverse effect upon hearing acuity.

As far as the cases of acute purulent otitis media are concerned, the average dose remains the same, but the number of treatments has been increased very slightly, as well as the time before the ear was dry.

The cases of chronic purulent otitis media show a slight increase in the number of treatments, an increase in the average dose of 12 r, and an increase in the duration of the discharge of almost two and a half days.

The only explanation we are able to give for the differences just mentioned is that,

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Table I

	Acute Catarrhal Otitis Media				Acute Purulent Otitis Media				Chronic Purulent Otitis Media			
	No. Cases	Average No. Treatments	Average Dose r	Drum Normal Days	No. Cases	Average No. Treatments	Average Dose r	Ear Dry Days	No. Cases	Average No. Treatments	Average Dose r	Ear Dry Days
First series	33	1	72.9	3.15	18	1.33	70.0	8.16	7	1.37	64.54	11.63
Second series	33	1	79.09	3.13	14	1.43	71.23	10.72	5	1.68	76.49	14.05
Total series	66	1	75.98	3.14	32	1.37	70.54	9.25	12	1.5	69.52	12.64

in such a small series of cases, a variation of this sort usually occurs. But in considering the series of cases as a whole, we are struck by the fact that the duration of any of the three types is much less than it would be without roentgen therapy. We wish to reiterate our statement that in this form of therapy, the otologist has a valuable means of treatment which should be more widely employed. We have yet to see any ill effect resulting from its use.

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ROENTGEN THERAPY OF DIPHTHERIA CARRIERS

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INTRODUCTION

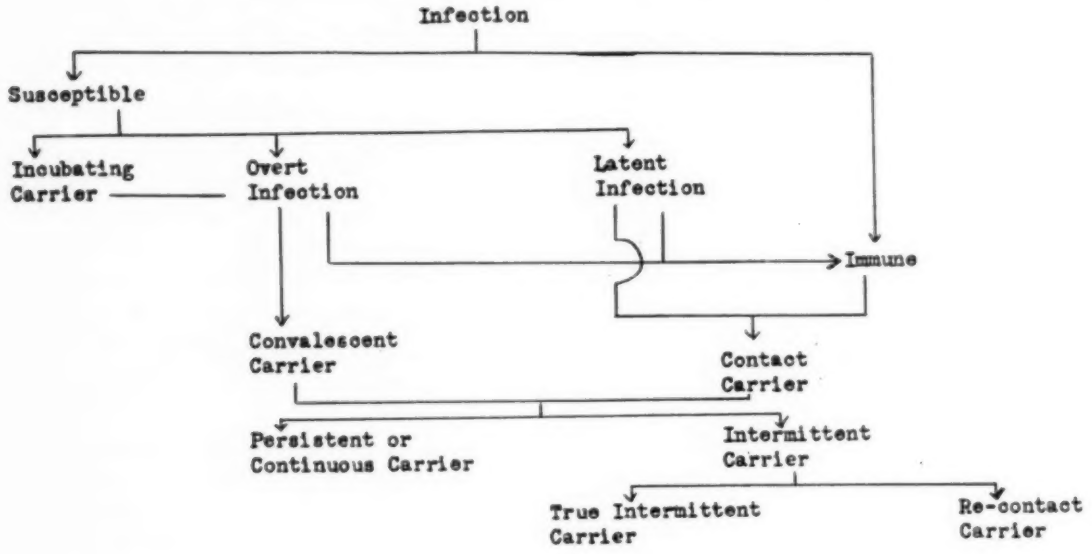
DIPHTHERIA carriers are a serious economic and public health problem to any community (to some, quite costly). The approach to the subject, as here presented, is intended as a rational and logical help. The cases presented are merely examples of the types, not an accumulation of data, and are intended to show the value of roentgen therapy.

Diphtheria carriers, as such, are stressed more than they should be, and not enough is said about how they should be treated, largely because no satisfactory means of therapy (aside from roentgen therapy) is available in a large percentage of cases.

DEFINITION

It is difficult to define a diphtheria carrier accurately. We believe no one would argue with the definition that a carrier is any person, not suffering with the disease, who harbors and disseminates the germs. However, this falls short of an accurate definition and leads us into many erroneous conclusions. Although there are several types of carriers, only a few are clinically important. The classification given by Mitman[12] shows this admirably:

CLASSIFICATION OF DIPHTHERIA CARRIERS (AFTER MITMAN)[12]



As can be seen, the persistent carrier and intermittent carrier are the most important. The other stages are really temporary and exist only during the time that the individual is developing immunity. From such an outline, one can readily see how these individuals might be falsely labelled carriers.

The methods of examination of a carrier are mostly laboratory procedures:

(1) Throat and nose swab
(2) Virulence test on positive culture
(3) The Schick test should also be included as a positive one indicates that the individual may be suffering from an overt or latent infection and is only a susceptible type of carrier. A susceptible carrier is the

type in which the Klebs-Loeffler bacilli disappear when the patient becomes immune (providing, of course, that there is no chronically diseased mucous membrane).

In addition to these laboratory examinations, the clinical examination of the nose and throat must be done, because a diseased mucous membrane is most important.

LOCATION OF INFECTION IN CARRIER STATE

The upper respiratory tract is the most common site of the bacilli in the carrier state. The chronic running ear is occasionally the site. Coachman[1] reported an eye carrier, but this and other sites are rare.

Harvey[6] and Kahn[10] found that the presence of an abnormal mucous membrane or physical abnormality in the nose, nasopharynx or throat was the main predisposing factor in starting or maintaining the carrier state. Harvey also found that the diphtheria bacilli were living only a saprophytic existence in the inflamed products of the mucous membrane.

The ability to cure a carrier has been, in the past, directly proportional to the accessibility of the organism. Throat carriers are easily cured, nose carriers are difficult to cure; the ear carrier has had no successful form of therapy. Since the use of roentgen therapy, however, all types of carriers have been successfully treated.

METHODS OF TREATMENT

Harvey's[6] exhaustive study of various types of treatment are summed up in his table of figures of all types of carriers:

Treatment	No. Cases	Total Cures per cent	Failures per cent
Vaccines	38	42.1	57.9
Nasal douching	55	78.0	22.2
Vaccines and nasal douching combined	7	57.1	42.9
Tonsillectomy and adenoidectomy	60	58.3	41.7

These figures are in accord with other workers. Hickey,[7] in his report, found 68 per cent cures with mercurochrome swabs.

The following results of roentgen therapy, published in this country and in Europe from 1922 to the present day, are by no means complete, but are representative of the results obtainable:

	Cases	Per cent
Hickey[7]	39	80
Streil[15]	150	97
Laquerriere, Kuentz and Roget[11]	17	100
Rogers[13]	18	100
Humphreys, Withers and Ranson[9]	39	90
Kahn[10]	185	100
Coachman[1]	24	95
	472	94.5

RATIONALE OF THERAPY

In treating diphtheria carriers, the aim is to elimate the organism. This is accomplished by: (1) restoring the diseased mucous membrane to normal; (2) immunizing the patient to the Klebs-Loeffler bacillus.

Removal of the diseased mucous membrane, or surgical correction of the condition causing pathologic changes, can accomplish much. However, due to the inaccessibility of some of the diseased tissues, surgical methods often fall short. In sinus operations, quite often, one pathological condition is merely substituted for another, as in the Caldwell-Luc operation of the antrum.

Rosenau[14] mentions the use of swabs, sprays, gargles, antiseptics, vaccine, toxin, antitoxin, as well as the overlaying the throats with cultures of staphylococci, but concludes: "We must acknowledge that all these measures often fail. The relief of bacillus carriers is one of the unsolved problems in preventive medicine!" This statement was made in spite of the fact that the value of roentgen therapy was established and reported as early as 1922. Even today, many refuse the benefit of roentgen therapy to carriers, either because of prejudice or lack of understanding of the value and the complete safety of the procedure. As will be shown later, the total dosage necessary is so small that there is not the slightest danger to the most protracted treatment that may be necessary.

As a good many carriers are due to the presence of a latent infection from a partial susceptibility, the simple procedure of immunization will eliminate a great many so-called carriers. Rosenau says that Klebs-Loeffler bacilli disappear in 50 per cent of cases by the time the local membrane has disappeared; in two months there are 5 per cent positive, and 2 per cent at three months; 1 per cent persists as chronic carriers. As can be seen by the chart of Mitman, the great majority of carrier types are due to susceptibility.

It has been shown by Desjardins[2,3] and Hodges and Berger[8] that the natural process of resistance to infection is accelerated by roentgen irradiation. The accumulation of old lymphocytes and leukocytes is destroyed and new cells allowed to enter the field. Phagocytosis by the reticulocytes is stimulated, probably by opsonins or other factors liberated by the destroyed lymphocytes. As a result, the hypertrophied and chronically inflamed lymphoid tissue is shrunken and dissolved.

Dubowyi and his associates[4,5] investigating the bacterial flora of the throat, found that following only one roentgen treatment, a large percentage of streptococcus and staphylococcus infection disappeared. Ullmann and Nuzum[16] obtained like results in a similar investigation.

TECHNIQUE OF TREATMENT

The nasopharynx is treated through two lateral fields up to 10 by 15 cm., depending upon the size of the patient. Treatments are given through each portal the same day, and repeated on alternate days until three are given. The series is repeated in one week if the cultures remain positive. If, after the second course, the culture remains positive, virulence tests should be done on the cultures. The series may be repeated the third time with complete safety, but neither in our cases nor in those reported was this necessary.

The following factors are used: 200 kv., 50 cm. target-skin distance, half-value layer = 1.00 mm. copper, 10 per cent of an erythema dose to each field. In those cases which received two courses, only 60 per cent of an erythema dose was given in all over a period of three or four weeks.

REPORT OF CASES

CASE I. L. C., aged fourteen, contact to brother who had diphtheria on July 15, 1939. Schick test negative in 1936. On August 9, 1939, cultures of her nose and throat were positive for Klebs-Loeffler bacilli. The virulence test was positive on August 15, 1939. On physical examination the throat appeared negative. Roentgen therapy was begun on August 2, 1939, and three treatments were given. Cultures became negative and after the three treatments remained so.

CASE II. D. C., aged ten, same family as Case I, contact to brother. Schick test negative in 1936. Nose and throat cultures were positive on August 9, 1939, and remained positive. On physical examination tonsils appeared slightly inflamed. Roentgen therapy was begun on August 23, 1939, and three treatments were given. Cultures remained positive, and a second course of roentgen therapy was repeated two weeks later. After the second series the cultures became negative.

CASE III. D. R., aged thirty-five, had a routine culture by the Health Department on June 24, 1939, which was positive. Virulence test was positive at the same time. This remained so in spite of other treatment and patient was referred for roentgen therapy. Schick test was negative. Physical examination revealed nose and throat to be negative. Roentgen therapy was begun on July 6, 1939, and three treatments were given. Cultures became negative and remained so after the treatments.

CASE IV. E. C., aged eight, had diphtheria on July 15, 1939. Nose and throat cultures were positive following the disease. Roentgen therapy was begun on September 15, 1939, and three treatments were given, but cultures remained positive. A second course of roentgen treatments was given and cultures became negative.

CASE V. M. L. A., aged eight, admitted on January 24, 1934. Five days before patient developed sore throat with high fever which persisted. Physical examination showed the cervical nodes to be enlarged and tender. The pharynx was red and swollen. Tonsils were large and injected and covered with tenacious gray membrane. Temperature 102.2° F. *Laboratory*

findings: Virulence test was positive on January 27, 1934. Urinalysis negative. Erythrocytes 6,000,000; leukocytes 13,800, with 81 per cent polymorphonuclears. On entrance 20,000 units of diphtheria antitoxin were given and the patient rapidly improved. However throat cultures remained positive. Roentgen therapy was begun on March 8, 1934, and five treatments were given. After three roentgen treatments, throat became negative and remained so.

Case vi. B. J. DeV., aged three, was admitted to the hospital on July 3, 1939, after a convulsion and vomiting. She had not been well for three days but, due to her mental deficiency, this was difficult to determine. Mother had had sore throat with fever for past week. *Physical examination:* Temperature 102° F. Entire throat inflamed. Tonsils enlarged, cryptic and covered with pus. No membrane seen. There was a spastic paraplegia of lower extremities, and patient appeared to be an idiot. *Laboratory findings:* Wassermann-Kahn negative; urinalysis negative. Erythrocytes 4,300,000, leukocytes 23,700, with 80 per cent polymorphonuclears. Electrocardiogram negative. On July 4, 1939, membrane appeared in throat. On July 5 cultures were positive for Klebs-Loeffler bacilli. Patient was given 20,000 units of diphtheria antitoxin and virulence test became negative at this time. Patient recovered in a few days and membrane disappeared. Patient remained well, but cultures continued positive and virulence test became positive. Roentgen therapy was started to throat and three treatments were given. Cultures became negative within two days and remained so.

Case vii. D. L. B., aged two years eight months, was admitted to the hospital on August 6, 1939, with a history of fever for two days. The day before entry throat was cultured and was found positive for Klebs-Loeffler bacilli. No sore throat was complained of. No immunization against diphtheria. *Physical examination:* Whitish-gray membrane on surface of tonsils. Twenty thousand units of diphtheria antitoxin were given intramuscularly in buttocks after negative sensitivity test. *Laboratory findings:* Urinalysis negative. Erythrocytes 5,100,000, leukocytes 32,400, with 89 per cent morphonuclears. Electrocardiogram negative. On August 14, 1939, tonsils were large with a small amount of exudate still present. Cultures and virulence test were still positive. On August 21 the exudate was gone, tonsils still inflamed and patient appeared well. On August 25 tonsils continued large and inflamed. On August 30, Ear, Nose and Throat Consultation advised tonsillectomy, which was performed on September 1. Roentgen therapy was begun on August 18 and three treatments were given. Throat cultures remained occasionally positive until tonsillectomy was done, after which they became negative. No virulence test was done after treatment.

Case viii. C. B., aged thirty-eight, was admitted on September 6, 1939. Patient was in jail and developed sore throat with malaise and vague pains in his body. He had difficulty in swallowing. A throat culture was taken on the third day and was positive for Klebs-Loeffler bacilli. He was sent to Isolation. *Past history:* For several years patient has suffered from attacks of sore throat with general malaise. At these times his tonsils would have a yellow exudate on them. These attacks would subside with the usual treatment. *Physical examination:* Tonsils present and small. Pharynx red and edematous. Examination otherwise negative. After a negative scratch test 10,000 units of diphtheria antitoxin were given intravenously without reaction. *Laboratory findings:* First positive culture on September 6, 1939; virulence test positive on September 11. Urinalysis negative. Wassermann and Kahn reactions negative. Erythrocytes 4,200,000, leukocytes 15,000, with 81 per cent polymorphonuclears and 12 per cent lymphocytes. On September 9 the throat was well, exudate had disappeared. Tonsils and pharynx were normal, no swelling; cultures were still positive. On September 25 patient complained of sore throat again and throat appeared red and swollen. Cultures still positive for Klebs-Loeffler bacilli. Roentgen therapy was started on September 27 and three treatments were given. Cultures became negative after first treatment and remained so.

Case ix. T. B., aged twenty-two, was admitted on September 8, 1939. He was in jail serving a thirty day sentence. His jail partner developed a sore throat and culture was positive for Klebs-Loeffler bacilli, and he was sent to the hospital. Patient had had diphtheria about twelve years ago. *Physical examination:* Dental caries. Tonsils present, small and cryptic and not inflamed. Remainder of physical examination negative. *Laboratory findings:* Schick test negative; nose and throat cultures positive for Klebs-Loeffler bacilli and virulence test was positive at the same time. Urinalysis negative. Wassermann reaction negative. Erythrocytes

4,460,000, leukocytes 80,800, with 86 per cent polymorphonuclears, 10 per cent lymphocytes and 4 per cent monocytes. Cultures remained positive and roentgen therapy was begun on September 27, 1939. Cultures still remained positive but virulence test became negative on October 5, 1939 and was also negative on October 21. Patient had tonsillectomy on day before dismissal and pathological report of tonsils was chronic tonsillitis.

CONCLUSIONS

(1) We have presented nine cases of diphtheria carriers of various types treated by roentgen irradiation, all of which were benefited by the treatment.

(2) All types should be treated by roentgen irradiation because of the possibility that one type can develop into another more chronic type. Immunization should be continued as at present, and its use spread to cover adults who are carriers and who are Schick positive.

(3) Mitman's outline clarifies the types of carriers, and cultures and Schick tests should be done simultaneously for more intelligent treatment. In Schick positive carriers (susceptible type) immunization should be combined with roentgen therapy.

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TUBERCULOSIS OF THE BREAST TREATED WITH ROENTGEN IRRADIATION*

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TUBERCULOSIS of the breast is a relatively rare disease. Lee and Floyd in 1934 found a total of 399 proved cases in the literature, of which 14 were in males. The disease usually occurs between the ages of twenty and forty.

Tuberculosis of the breast is usually unilateral. Of the 399 cases collected by Lee and Floyd, the disease was bilateral in only 13. Tuberculosis of the breast is considered to be primary when there is no evidence of the disease in any other part of the body. In the 399 cases mentioned above, 234 were classified as primary. Tuberculosis may attack the axillary lymph nodes and secondarily invade the breast. This may have been the case in the patient to be presented here. The axillary lymph nodes are said to be involved in over 50 per cent of the cases.

DIAGNOSIS

The most frequent symptom is a painless lump in the breast. When seen for the first time, the differential diagnosis from cancer of the breast may be impossible. The early involvement of axillary lymph nodes in tuberculosis is of no aid in the diagnosis. However, the disease progresses more rapidly than cancer. Early ulceration with sinus formation is of great differential diagnostic value, since this occurs very frequently in tuberculosis of the breast. The diagnosis should be made as soon as possible without waiting for the skin ulceration. Unless this occurs, only histological examination of the removed specimen can give the final diagnosis. One must also realize that tuberculosis and cancer may be coexistent in the same tumor, or cancer may be present in the breast and tuberculosis in the axillary lymph nodes. The tubercle bacillus has been identified in only a few cases of tuberculosis of the breast, and in all these the bovine type was found. However, the number of instances in which cultures of the bacilli were made is as yet too small to justify any definite conclusion. In cases of breast abscesses which do not heal within a few weeks, even though bacteriological study is negative for the tubercle bacillus, a biopsy of the abscess wall is advisable to rule out tuberculosis.

PATHOLOGY

Berger and Mandelbaum divide the pathologic changes of tuberculosis of the breast into three groups.

1. Nodular type (discrete, disseminated and confluent). The discrete form is the most frequent. The tubercle is usually situated in the connective tissue, rarely in the duct or periductal tissue. The acini are destroyed by reactionary lymphatic infiltration. The tubercle enlarges to form a mass. Caseation takes place, and if suppuration occurs, sinuses are formed. Daughter tubercles form at the periphery of the area in the disseminated type. In the confluent form, liquefaction necrosis takes place in several of the tubercles and these caseous suppurative masses communicate with each other.

2. Sclerosing type. Usually in old persons. The breast becomes small, hard and shrunken.

3. Atypical: (*a*) obliterating tuberculous mastitis (obliteration of the ducts); (*b*) intraglandular cold abscess represents an advanced stage of the confluent type.

TREATMENT

Medical. Berger and Mandelbaum believe that tuberculin injections produced considerable improvement in one patient with tuberculosis of the breast. These authors state that "any form of medical treatment

* From the Tumor and Radiation Therapy Service, Morrisania City Hospital, New York.

that offers the possibility of cure without subjecting a young woman to the humiliation consequent to the loss of a breast is deserving of a thorough trial."

Surgical. Excision and drainage of a tuberculous abscess usually results in failure of its cure. In the discrete nodular type a local excision may result in complete healing.[7,9] However, local excision of the segment of the breast involved is frequently not curative because the sinus tracts are tortuous and one is apt to leave behind an area of disease in which another abscess will develop later.[1] Some advocate simple mastectomy in all cases, especially if skin ulceration is present. If the axillary lymph nodes are involved, an axillary dissection should be done at the same time. It has also been suggested that the breast alone be operated upon and the axillary lymph nodes be treated with radiation.

Radiotherapy. The well known success with radiation therapy in the treatment of tuberculosis of peripheral lymph nodes should suggest the use of this form of therapy in tuberculosis of the breast. The reasons for the use of roentgen therapy are as follows: (1) there is no mutilation and psychic trauma such as accompanies mastectomy; (2) there is no mortality, and (3) there is a high cure rate.

Search of the literature has revealed only one publication on irradiation of tuberculosis of the breast. Glauner, who is the author of this article, reported 3 cases treated only with roentgen rays and all were well for periods varying from three to eighteen months after the commencement of the therapy. He strongly advises that roentgen therapy should always be tried before surgery. Holfelder, in discussion of above publication, added one other case of tuberculosis of the breast which was irradiated at his Institute; this patient was well for three years. Goldberg has recently also treated such a case. Radiation therapy was given to a lump in the breast prior to an intended biopsy. The mass disappeared but an area of redness and finally sinus formation resulted. Guinea pig inoculation of the discharge from the sinus revealed the presence of tubercle bacilli. Radiation therapy was given to the sinus and a cure was obtained.

TABLE I

AREAS IRRADIATED AND DOSAGE ADMINISTERED TO EACH

Dates	Field Irradiated	Size of Field	Total Dose in Roentgens
10/ 2 to 12/12/39	Left breast, mesial oblique	17×15 cm.	500
10/ 2 to 12/12/39	Left breast, lateral oblique	17×15 cm.	500
10/13 to 12/12/39	Left axilla, anterior	10×15 cm.	800
2/19 to 3/20/40	Left lateral breast and axilla	17×17 cm.	500
5/24 to 6/24/40	Left axilla anterior, lateral oblique	8×10 cm.	300
5/24 to 6/24/40	Left axilla anterior, mesial oblique	8×10 cm.	350
8/12 to 8/27/40	Left axilla direct	8×10 cm.	400

Technique of Roentgen Therapy. Glauner used 110 kv., 0.2 mm. Cu, as well as 180 kv., 0.5 mm. Cu filter with doses of 90 to 150 r (measured in air), every two to three weeks for three to twelve treatments until the disease had disappeared. Holfelder employed 180 kv., 0.5 mm. Cu filter, with doses of 250 to 400 r (measured in air), every week or so.

REPORT OF CASE

M. A., unmarried female, negress, aged twenty-eight, first noted a left axillary swelling in May, 1939. This drained spontaneously within a few days. Several days later, the left breast began to enlarge and became hot and tender to the touch. She entered another institution on June 24, where an incision and drainage of the swelling in the left breast was done. Smears from the pus obtained showed gram-

positive cocci and *Staphylococcus aureus*. Culture from the pus in the left axillary nodes revealed *B. coli*. Blood culture was negative, as was also roentgen examination of the chest. Patient was discharged from that hospital after a stay of one month. She was re-admitted two and one-half months later, September 8, 1939, because the abscesses in breast and axilla did not heal. A wide incision was made over an abscess in the left breast and 500 cc. of creamy pus were evacuated. At the same time, a biopsy

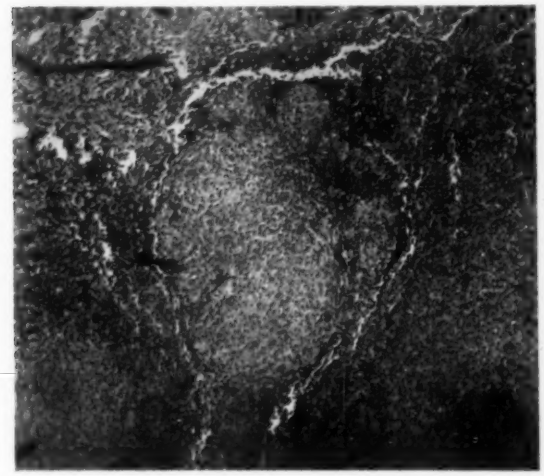

Fig. 1. Low power magnification of tissue removed at biopsy showing tubercle formation.

was taken from the wall of the abscess. A culture of the pus showed *Staphylococcus albus*. The histological report of the removed tissue was "tuberculosis." This diagnosis was confirmed by our Pathology Department (Fig. 1.) The patient was then referred for radiation therapy.

The patient was first seen in our Hospital on October 2, 1939, and the following note was made: "There are two large longitudinal incisions in the upper outer quadrant of the left breast. The wounds are wide open and present clean granulating surfaces. There is considerable induration surrounding these wounds. A large mass of firm lymph nodes, 10 cm. in diameter, is present in the left anterior axilla, extending into the infraclavicular area (Fig. 2). The supraclavicular area is normal. The right breast and regional lymphatics are normal." Roentgen examination of the chest showed no evidence of any abnormality.

Radiation therapy was begun on October 2,

Fig. 2. Appearance of lesion in breast prior to radiation therapy. The extent of lymph node enlargement in axilla is outlined.

1939 (Table 1). The physical factors employed were: 200 kv., 20 ma., 0.5 mm. Cu plus 1.0 mm. Al filter (half-value layer 0.89 mm. Cu), and 50 cm. focal-skin distance. The entire left breast was irradiated using two tangential fields, me-

Fig. 3. Appearance of breast in March, 1941, eight months after last roentgen treatment.

sial and lateral, each measuring 17 by 15 cm. The axillary lymph nodes were treated at first by using 10 by 15 cm. portals, but as the nodes decreased in size, these were reduced to 8 by 10 cm. The dose per field was 75 to 100 r, measured in air, at weekly intervals.

Course. On October 13, 1939, the wounds in the breast looked much cleaner. On February 19, 1940, four months after treatment was started, the axillary lymph nodes were barely palpable but there was still some induration in the left breast. There was a recurrent draining sinus, 1.0 cm. in diameter, in left mid-axilla noted on April 11. This healed three months later but recurred again in one month. After additional irradiation to this region, this sinus finally healed at the end of two more weeks. The last roentgen treatment was given in August, 1940. The patient has been well and free of disease to date, May, 1941, a period of nine months since last treatment and nineteen months since treatment was begun (Fig. 3).*

SUMMARY

Tuberculosis of the breast occurs infrequently and then usually in women between the ages of twenty to forty.

The differential diagnosis from cancer of the breast may be impossible when ulceration and sinus formation have not yet occurred.

Biopsy of the abscess wall is advisable in cases of breast abscesses which do not heal promptly under conventional therapy.

Since local excision of the involved segment of the breast is reported to be unsuccessful in many cases, simple mastectomy must be done for cure.

Roentgen therapy is recommended in tuberculosis of the breast as a conservative and successful form of treatment.

A case of tuberculosis of the breast is presented which was treated with roentgen irradiation and is now free of disease for nine months since last treatment.

* The patient was last seen in March, 1942, twenty-nine months after treatment, and is still perfectly well.

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DIRECT OR INDIRECT ACTION OF ROENTGEN RAYS ON THE BRAIN*†

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IT IS WELL established that the adult nervous system is very radioresistant. However, from animal experiments the possibility of injury to nerve tissue can now be entertained.[1] Whether the changes observed in the brains of irradiated animals and human beings are the result of a direct effect of irradiation on the nervous tissue or whether they are secondary effects due to a primary injury of the blood vessels is still an open question.

Examination of the brains of irradiated goldfish by Davison and Ellinger[2] revealed definite changes predominantly in the medulla oblongata. The severity of these changes was dependent on the dose used.

In previous investigations[3] it has been demonstrated that for the total body irradiation of goldfish there is a definite threshold dose of 1,500 r, measured in air, for the lethal effect of roentgen rays of half-value layer 0.233 to 0.9 mm. Cu. Increase of the dose up to 10,000 r, measured in air, did not influence the mortality curve. The histological examination of the brains, however, revealed an increase in the histopathological changes, especially in the medulla oblongata (Figs. 1 and 2) when these doses were given. It is noteworthy that the fish irradiated with 10,000 r died on the thirteenth day after irradiation and that the control fish, irradiated with the threshold dose of 1,500 r, were killed on the fifteenth day after exposure. The survival time is, therefore, approximately equal. This is of importance since changes in the central nervous system following irradiation require a considerable time to develop. It is important, furthermore, that the changes in the blood vessels did not differ greatly when either dose was used (Figs. 1

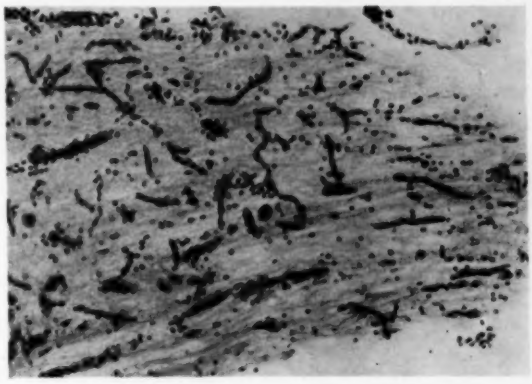

FIG. 2. Medulla oblongata. Goldfish irradiated with 1,500 r, measured in air. Pigmentation appeared on the eleventh day after exposure. Fish killed on the fifteenth day after exposure for histological examination. Medulla oblongata stained with cresyl violet. Magnification 240×. As compared with Figure 1 there is only a slight increase in glia nuclei and the nerve cells are only diminished in number. Marked congestion of the blood vessels. Some extravasation of red blood cells.

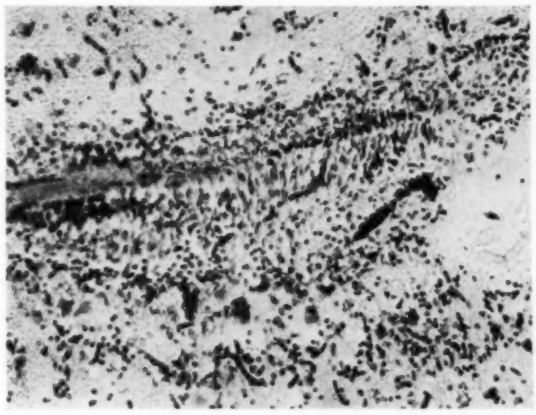

FIG. 1. Medulla oblongata. Goldfish irradiated with 10,000 r, measured in air. Pigmentation appeared on the sixth day after exposure. The fish died on the thirteenth day after exposure. Medulla oblongata stained with cresyl violet. Magnification 240×. Gliotic process. Note the parallel arrangement of glia fibers, disappearance of the nerve cells, congestion and slight proliferation of blood vessels.

* From the Radiotherapy Department and the Neuropathological Laboratory, Montefiore Hospital for Chronic Diseases, New York.
† Aided by a grant from the Emergency Committee in Aid of Displaced Foreign Physicians.

and 2). *The assumption seems, therefore, justified that the differences in the effects of increasing doses of roentgen rays on the brain is due to direct action of the radiation on the nerve cells.* The marked radioresistance of the nerve cells accounts for the longer period before injury becomes detectable. In consequence, the damage done to more vulnerable tissues, e.g. blood vessels, dominates the histological picture. But this does not necessarily imply that the injury to the nerve cells is secondary to such changes.

The brain of the goldfish is an excellent medium for the study of the effects of roentgen rays for the following reasons: (1) Such studies do not have to take into consideration the absorption of the radiation in the calvarium; (2) the experiments can be carried out without opening the skull, and (3) the data obtained can be easily related to the mortality curve of the goldfish, an ideal condition in experimental radiation therapy.

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AN EXPOSURE METER FOR ROENTGENOGRAPHY*

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THE importance of correct exposure in the production of diagnostically excellent roentgenograms is recognized by everyone. For many years it has been almost the universal practice to estimate roentgenographic exposures from the thicknesses of the anatomical structures under consideration. This method is frequently satisfactory for examinations of normal tissues but often unreliable when pathological processes are present. The reason for this is evident. Roentgenographic exposure is influenced not only by the thickness of a body but also by its density.

If the effective intensity of the radiation to be received by a roentgenographic film is precisely known, the conditions for correct exposure can be determined from a simple expression relating radiation intensity and exposure time. Several instruments designed to measure roentgen intensities and often called exposure meters or photometers have been produced commercially on the European continent. Most notable are the devices developed by Franke[2,3] and Pape.[6,7,8] These are not used extensively, however, because of their low sensitivity and critical operating characteristics. The exposure meter to be described presently overcomes these difficulties and appears to be a valuable aid to roentgenographic technique.

The instrument consists essentially of a photoelectric cell whose surface is coated with a fluorescent material, a vacuum tube amplifier and a microammeter with which the amplified photocell current is recorded. The procedure to be followed in using the device is extremely simple. The photocell is placed beneath an anatomical structure to be roentgenographed, a roentgen beam projected through the tissues, the resulting deflection of the microammeter observed and the proper exposure determined from a graph expressing exposure times in terms of exposure meter readings. The photocell is then removed from beneath the subject, a film inserted in the cassette holder and the exposure made with a roentgen beam identical to that employed in making the photometric measurement. Because the response of the instrument is affected by both the thickness and density of the tissues, exposures are consistently correct.

FUNDAMENTAL BASIS OF PHOTOMETRY†

The density (blackening) of an exposed and processed photographic film is dependent on several complex variables. These include the exposure received by the film, the sensitivity of the photographic emulsion and several factors related to the process of development.

Photographic exposure is defined as the product of the average photographic flux density,‡ G, and the exposure time, t:

$$E = Gt \qquad (1)$$

As this definition indicates, exposure is not only a function of the quantity but also of the quality or spectral distribution of the radiation falling on the film.

In general, the exposure required to produce a particular film density is inversely proportional to the sensitivity of the film; that is,

$$E = \frac{a}{s} \qquad (2)$$

where s is the film sensitivity and a is a constant whose value is a function of the film density. When equations (1) and (2) are combined,

$$Gts = a \qquad (3)$$

† The principles outlined herein are equally applicable to roentgenographic and photographic films.

‡ A discussion of the significance of the term, photographic flux density and its relationship to radiation intensity has been published by Weyl, Warren and O'Neill.[9]

* From the Division of Roentgenology of the University of Chicago. Read before the Chicago Roentgen Society, April 10, 1941.

The reciprocal relationship between G and t expressed in equation (3) is not maintained when the photographic flux density is either very small or very great.[4] Although the literature is not complete on this point it would appear that the effect is more pronounced with films exposed to visible light than to roentgen radiation.[1] Experimental evidence indicates that equation (3) to be more general should be written

$$Gt^p s = a \tag{4}$$

where the exponent, p, is a function of the intensity and spectral distribution of the radiation. Within the range of intensities employed in roentgenography the value of p for films exposed to roentgen rays directly is very nearly 1.0; it is usually somewhat less for films exposed with intensifying screens.

The density of a particular area of a photographic film may vary within wide limits. However, if a series of correctly exposed negatives is examined it will be observed that the average density of the significant portion of any one film is, almost without exception, equal to that of any other. Therefore, in the majority of cases, correctly exposed films can be represented by a single value of a in equation (4). If this value is a_e the conditions for correct photographic exposure are defined by the equation,

$$Gt^p s = a_e \tag{5}$$

When light impinges on the sensitive surface of an exposure meter or photometer, the resulting deflection, M, is a function of the photometric flux density, P:

$$M = f(P) \tag{6}$$

The derivations of photometric flux density and of photographic flux density are analogous. The relationship between the two terms is expressed by the equation,

$$G = hP \tag{7}$$

where h is a function of the spectral distribution of the radiation and of the sensitivity of the exposure meter. Equation (5) therefore becomes

$$Pt^p hs = a_e \tag{8}$$

For simplicity the product, hs, may be represented by the factor, s_e, called the film speed factor from its relationship to film sensitivity: thus,

$$Pt^p s_e = a_e \tag{9}$$

Equation (9) is of fundamental importance. With it photographic exposure times can be calculated from photometric observations for any film whose speed factor is known.

When the relative spectral sensitivity of an exposure meter coincides with that of a photographic film, equation (7) reduces to a simple proportionality and s_e ceases to be a function of radiation quality. In roentgen photometry, it is desirable to take advantage of this fact as far as it is possible to do so because the quality of the radiation with which roentgenographic films are exposed varies within wide limits and depends not only on the characteristics of the primary roentgen beam but also on the thickness and density of the tissues through which the radiation passes. The densities of roentgenographic films are produced either by roentgen rays directly or indirectly through the action of intensifying screens. In the former case the wave length of the effective radiation ranges from 0.015 to 0.150 millimicron; in the latter from 360 to 500 millimicrons. With present equipment an exposure meter whose relative spectral sensitivity coincides with those of roentgenographic films in both ranges is not feasible. It has been possible, however, to develop an instrument that closely approaches this ideal.

DESIGN AND CONSTRUCTION

The exposure meter (Fig. 1) is similar in basic design to the photoelectric photometers that have proved so reliable in photography. A photoemissive cell whose surface is coated with crystals of calcium tungstate, the material employed in the great majority of commercial intensifying screens, conducts a small electric current in response to the fluorescent radiation produced by an incident roentgen beam. This current is amplified many times by a D. C.

amplifier and recorded by a sensitive microammeter.

The nucleus of the instrument is the recently developed R.C.A. vacuum type 929 phototube, a photoemissive cell which, unlike other similar devices, is sufficiently sensitive to detect the extremely small quantities of radiation encountered in clinical roentgenography without employing vacuum tube amplification so high as to be unstable. In addition, its relative spectral sensitivity, shown graphically with that of Eastman blue brand ultra-speed roentgen

Fig. 1. The exposure meter.

film in Figure 2, approaches those of commercial roentgenographic films in the region of the spectrum in which calcium tungstate fluoresces. It is clear, therefore, that the tube is an ideal photometric radiation detector when films are to be exposed with intensifying screens; its performance with films exposed to roentgen rays directly also has been found to be very satisfactory.

The amplifier, shown schematically in Figure 3, is a single stage, twin triode type similar to that described by Nottingham.[5] Two R.C.A. type 6J7 radiotrons, V_1 and V_2, connected as triodes are arranged in a Wheatstone bridge circuit in which the plate resistances of the tubes occupy two of the arms and R_{b1} and R_{b2}, 15,000 ohm resistors, occupy the remaining two. The phototube current flows through the 500

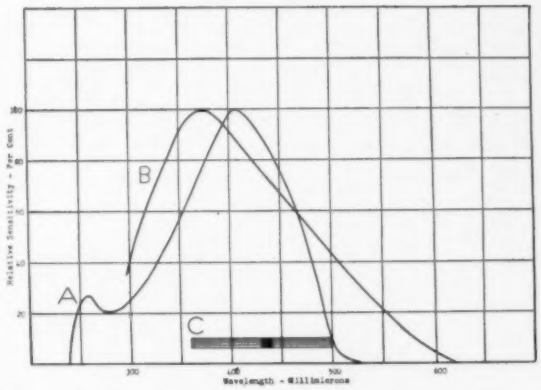

Fig. 2.* Spectral sensitivity characteristics: *A*, Eastman blue brand ultra-speed roentgen film; *B*, R.C.A. 929 phototube; *C*, region of the spectrum in which calcium tungstate fluoresces.

megohm resistor, R_{c1}; the potential developed across it is applied directly to the control grid of V_1. A 0–250 microammeter, G, in the bridge circuit measures the amplified phototube current.

Electric power for the amplifier is obtained from 115 volt, 60 cycle A.C. lines. Direct current potentials are provided by a condenser filtered, full wave rectifier circuit supplied by a 430 volt center-tapped winding on the transformer, T_1. The rectifier, V_3, is the R.C.A. type 6ZY5-G radiotron; the filter, C_4, a 16 microfarad, 350 volt condenser. Phototube and triode grid

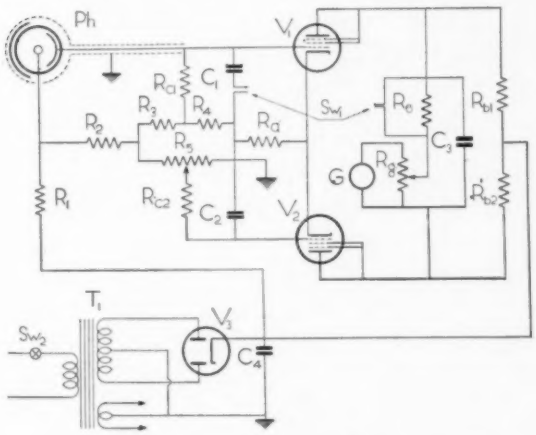

Fig. 3. Schematic diagram of the exposure meter circuit.

* Published with the permission of the Eastman Kodak Company and the Radio Corporation of America. Curve A was prepared from data furnished by Dr. Julian H. Webb and is typical of the film as of November, 1940.

potentials are derived from the resistor network, R_1, a 0.2 megohm resistor, R_2, a 15,000 ohm resistor, R_3 and R_4, 250 ohm resistors, R_5, a 500 ohm potentiometer and R_6, a 600 ohm resistor. Filament potentials for the amplifier and rectifier tubes are furnished by a 6.3 volt winding on T_1.

The potentiometer, R_5, maintains the balance of the Wheatstone bridge circuit by its control of the grid potential of the triode V_2; a 2,000 ohm potentiometer, R_g, controls amplifier sensitivity.

R_{c2}, a 500 megohm resistor, balances R_{c1} in the amplifier grid circuit. C_2, a 0.001 microfarad condenser in parallel with R_{c2},

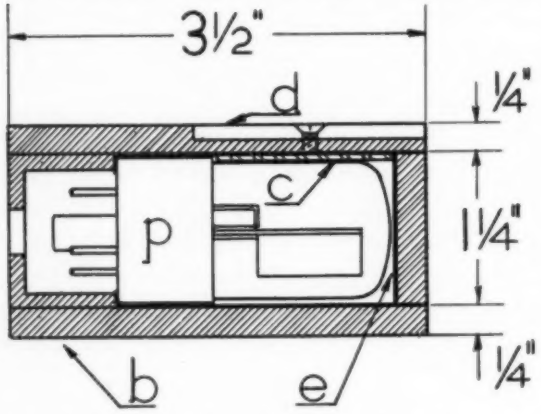

FIG. 4. Longitudinal section of the radiation detector. *b*, bakelite case; *c*, intensifying screen; *d*, stationary grid; *e*, aluminum foil; *p*, phototube.

forms a low resistance pathway for undesirable A.C. currents induced in the grid circuit of V_2 by the triode's interelectrode capacitance. Sufficient capacity in parallel with R_{c1} is provided by the distributed capacity of a cable connecting the phototube, Ph, and the triode, V_1.

C_1, a 0.002 microfarad condenser, C_3, a 100 microfarad condenser, and R_6, a 5,000 ohm resistor impart a ballistic-like action to the microammeter when photometric procedure demands the employment of short exposure times. Their inclusion in the circuit is effected by the double pole, single throw switch, Sw_1; it is to be noted that the switch is arranged so that one set of contacts is closed when the other is open. It is not to be inferred that these circuit elements convert the microammeter into a ballistic instrument. Meter deflections are proportional to radiation intensities only when a fixed exposure time is used.

The exposure meter is assembled in two sections; one serves as the radiation detector and the other as the control unit. The phototube is mounted with a strip of Patterson Hi-Speed intensifying screen, $1\frac{1}{2}$ inches square, in a light-proof bakelite case, $3\frac{1}{2}$ inches long, 2 inches wide and $1\frac{3}{4}$ inches high. The walls of the enclosure are lined with aluminum foil to shield the phototube electrostatically. An easily detachable 5:1 stationary grid, 2 inches square, is also incorporated in the case directly above the sensitive surface of the phototube. The structural details of the assembly are shown in Figure 4. The remaining components of the instrument are housed in a steel cabinet, 8 by 8 by 8 inches. The microammeter, a 4 inch square case model with knife-edge pointer and a 0–100 scale, the potentiometers, R_5 and R_g, and the switch, Sw_1, are mounted on the sloping front panel. The two units are connected by a special two wire cable in which the lead connecting the cathode of the phototube to the control grid of the triode, V_1, is shielded microphone cord; the capacity between the shield and the enclosed wire is 25 micromicrofarads per foot. To prevent the capacity in the amplifier grid circuit from becoming excessively large and thereby slowing the action of the microammeter undesirably, the length of the cable is limited to approximately 20 feet.

When the construction of the exposure meter is completed, it is advisable to seal the connections at the base of the phototube with paraffin to insure the satisfactory operation of the instrument in humid weather. Also, before the exposure meter is used for the first time it is necessary to heat the filaments of the amplifier tubes at their rated potentials (6.3 volts) for twenty-four hours. Gas absorbed on the surfaces of the cathodes of new tubes is often sufficient to cause erratic performance of high grid resistance circuits. This short heating per-

manently liberates the gas and eliminates any undesirable effects that might arise from its presence.

CALIBRATION AND OPERATION

The response of the exposure meter to incident radiation is linear over a range of intensities considerably greater than that encountered in clinical roentgenography:

$$M = kSP \qquad (10)$$

where S is the sensitivity of the instrument and k is a constant. This relation was derived from readings observed when the radiation detector was exposed to a carefully diaphragmed roentgen beam of constant quality at various traget-detector distances. Corrections for the effects of air absorption were applied to the observations.

When P is eliminated from equations (9) and (10),

$$Mt^p s_e = cS \qquad (11)$$

where $c = a_r k$. Equation (11) defines the conditions for correct roentgenographic exposure in terms of exposure meter readings.

The calibration of the instrument is extremely simple. The control unit is placed beside the roentgen control stand, plugged into a convenient A.C. outlet and the switch, Sw_2, closed. After a few minutes have elapsed to permit the amplifier tubes to become heated the sensitivity control, R_s, is turned to its maximum setting and the balancing control, R_b, rotated until the microammeter is brought accurately to its zero position. A subject is then placed on the roentgenographic table and the roentgen tube centered over an anatomical structure whose thickness is approximately 20 cm.; the 90° posteroanterior projection of the skull has been found satisfactory. The radiation detector with its stationary grid attached is then placed beneath the subject directly below the target of the roentgen tube. When the roentgen machine has been adjusted to a suitable kilovoltage and milliamperage (e.g., 80 kv. (peak), 30 ma.) an exposure of one second duration is made and the deflection of the microammeter observed. With an identical roentgen beam, the exposure time required to produce a diagnostically excellent roentgenogram of the structure is determined by a method of trial and error for the film-screen combination for which the exposure meter is to be calibrated. The Potter-Bucky grid is employed in making the various trial exposures. A graph expressing correct roentgenographic exposure times in terms of exposure meter readings is then prepared from equation (11) by plotting on logarithmic paper a point representing the observed exposure meter reading and the corresponding exposure time of the test roentgenogram and by drawing through the point a straight line at an angle of 135° with the M-axis; a typical calibration curve is shown in Figure 5.

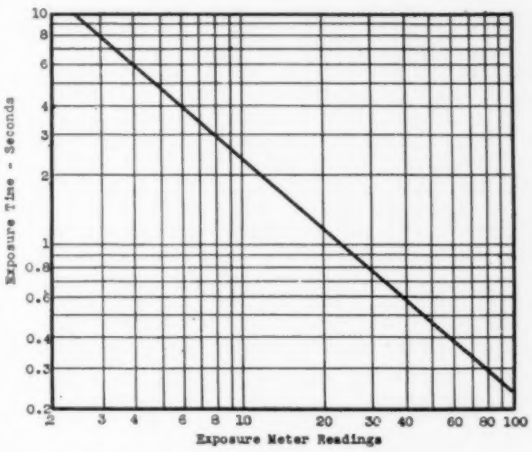

Fig. 5. A typical calibration curve.

To permit the use of the exposure meter with any roentgenographic film or film-screen combination any one of three procedures is followed: (1) A separate calibration curve is prepared for each of the various films and film-screen combinations that are to be used. (2) A calibration curve is prepared on any one film or film-screen combination; roentgenographic exposure times, t, are then calculated from the formula,

$$t = \frac{s_{e1} t_1}{s_e} \qquad (12)$$

where s_{e1} and s_e are the speed factors respectively of the film with which the calibration curve was prepared and of the film with which the roentgenogram is to be made and t_1 is the exposure time determined from the calibration curve. (3) Procedure (2) is carried out and the sensitivity control R_g, of the exposure meter calibrated in terms of s_e so that

$$S = gs_e \qquad (13)$$

where g is a constant. From equations (11) and (13),

$$Mt^p = cg \qquad (14)$$

Thus, when R_g is adjusted to the proper speed factor setting of the particular film or film-screen combination being used, determinations of exposure time are made from a single calibration curve without the use of mathematical formulae.

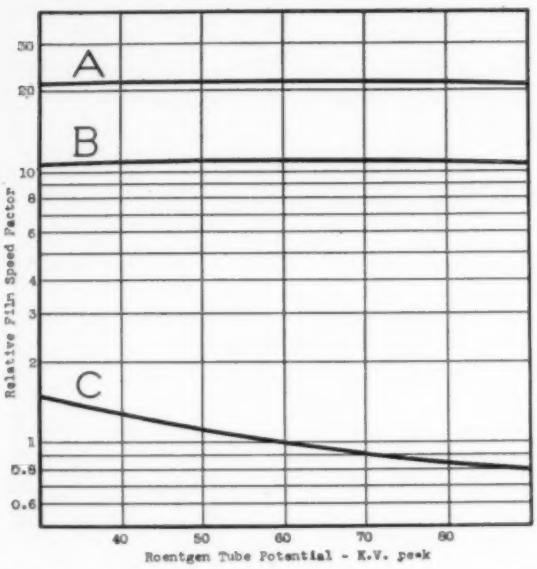

FIG. 6. Relative speed factors of a representative roentgenographic film; *A*, with so-called ultra-speed intensifying screens; *B*, with so-called high definition intensifying screens; *C*, without intensifying screens.

Before details of a method for determining the values of film speed factors are discussed, the results of a study investigating the relation between s_e and radiation quality will be presented. Several commercial roentgenographic films, used with and without intensifying screens, were exposed to radiation from a four valve, full wave rectified roentgen generator, employing potentials ranging from 30 to 90 kv. (peak), and developed simultaneously under constant agitation in a pictol-hydroquinone developer for five minutes at a temperature of 65° F. Their densities were measured with a photoelectric densitometer; after corrections for fog were applied, sensitometric curves were prepared and the exposures (Mt) required to produce a film density of 1.0 were determined. The relative speed factors were then derived from equation (11). In the calculations a value of 1.0 was assigned to the exponent, p. The procedure was repeated three times and the determinations averaged. A summary of the results of the investigation is given in Figure 6. Curve A is the relative speed factor of a representative roentgenographic film used with so-called ultra-speed intensifying screens; curves B and C are the relative speed factors of the same film used with so-called high definition intensifying screens and without intensifying screens respectively. It is seen that the speed factors of the film, when used with intensifying screens, are essentially independent of the spectral distribution of the radiation; that of the film exposed to roentgen rays directly diminishes as the average wave length of the radiation decreases. With minor modifications, the shape of these curves is characteristic of all films and film-screen combinations investigated.

To evaluate the speed factors of a number of films and film-screen combinations, the exposure times required to produce satisfactory and closely similar roentgenograms of a test anatomical structure (e.g. the hand) are determined by a method of trial and error. Exposures are made with a roentgen beam of constant quantity and quality and the various films developed simultaneously under typical roentgenographic conditions. The various values of s_e are given by the reciprocals of the exposure times (see equation 11). The speed factors of film-screen combinations are essentially independent of the quality of the roentgen radiation; those of films ex-

posed to roentgen rays directly must be multiplied by a correction factor, derived from curve C of Figure 6, when the films are to be exposed to radiation whose quality is different from that employed in determining the values of s_e. Because of their dependence on radiation quality, these factors must be further corrected for the thickness of tissue through which the radiation passes before it falls on the film, in accordance with the photometric principles discussed earlier in the paper. Correction factors for tissue thicknesses ranging from 0 to 15 cm. are shown in Figure 7. The graph applies only to films used without intensifying screens and exposed through the Potter-Bucky grid; no corrections are necessary for other roentgenographic techniques.

Figure 7 was prepared by evaluating the speed factors of films exposed through paper phantoms, 22 by 28 cm. in surface area, ranging in thickness from 0 to 15 cm. and having a density slightly less than that of water; determinations were made with and without the Potter-Bucky grid. Radiation was furnished by a four valve, full wave rectified roentgen generator employing potentials ranging from 30 to 90 kv. (peak). Correction factors were calculated from the formula

$$F = \frac{s_{ex}}{s_{eo}} \qquad (15)$$

where s_{ex} and s_{eo} are the speed factors of films exposed to radiation identical in quality at the reontgen tube but having passed through phantom thickness of x and o cm. respectively. Curiously the value of F was found to be essentially 1.0 for all phantom thicknesses when the exposures were made without a grid. Apparently scattered radiation was sufficient to compensate for the loss of the longer wave lengths of the primary roentgen beam removed by the phantom. When the grid was used, however, scattered radiation was prevented from falling on the film and there was a gradual reduction in the values of the speed factors as the phantom increased in thickness. In the range of thicknesses shown, Figure 7 was found to be independent of the quality of the primary roentgen beam.

To calibrate the exposure meter for chest roentgenography or for other techniques in

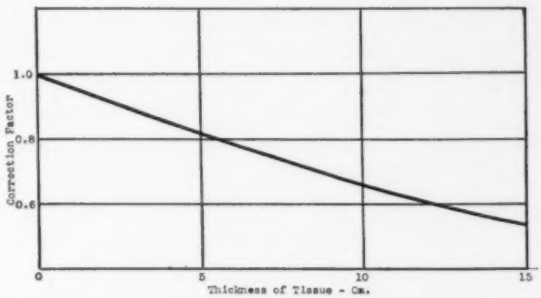

Fig. 7. Graph for obtaining the film speed correction factors of films exposed without intensifying screen through a Potter-Bucky grid and various thicknesses of tissue.

which it is not feasible to use exposure times as great as one second, the same general procedure previously described is followed with the exception that the switch, Sw_1, is thrown to the position in which the microammeter has a ballistic-like action. If the films are exposed with a Potter-Bucky grid, exposure meter readings are made with the radiation detector's attachable grid in place; otherwise the grid is removed. Observations are taken with exposure times of 0.1 second or some other convenient short interval.

When the various calibrations are completed the exposure meter is ready for routine clinical use. The procedure to be followed is similar to that employed in calibrating the instrument. The balancing control, R_5, is rotated until the microammeter is brought accurately to its zero position and the sensitivity control, R_g, adjusted to the proper speed factor setting for the particular film or film-screen combination with which the roentgenogram is to be made. The radiation detector is placed carefully beneath the anatomical structure to be filmed and the roentgen tube centered properly above. The positioning of the radiation detector usually is not critical. After a few trials, the technique of centering the device is quickly acquired. The roentgen machine is then adjusted to suit-

able kilovoltage and milliamperage settings an exposure of approximately one second made, the exposure meter reading observed and the roentgenographic exposure time determined from the calibration curve. If the film is to be made with short exposure time technique, the observation is made with the switch, Sw_1, in the so-called ballistic position and with the same exposure time as that employed in preparing the calibration curve. If the roentgenogram is to be made through the Potter-Bucky grid, the exposure meter reading is taken with the radiation detector's attachable grid in place; otherwise the grid is not used. When the determination is completed the radiation detector is removed from beneath the patient, a cassette placed in the film holder and the proper exposure made with a roentgen beam identical to that employed in making the photometric measurement.

The exposure meter is extremely flexible in its operation. The calibration is independent of the target-film distance so long as measurements are made with the radiation detector occupying the same position as the film. The instrument, therefore, is valuable in portable as well as routine clinical work.

COMMENT

For many years roentgenologists have recognized the need of a simple, convenient instrument whereby roentgenographic exposures might be calculated with consistent accuracy regardless of the conditions at hand. To fulfill this requirement to some extent, at least, the exposure meter described in the foregoing paragraphs has been developed; its performance in routine clinical work has been very encouraging. As time goes on it is hoped that many improvements and modifications designed to serve particular applications will be devised. It has been found recently, for example, that it is advantageous to mount the amplifier triode, V_1, in the case of the radiation detector or in a separate unit nearby when the exposure meter is to be installed as an integral part of the roentgen machine. Because the cable connecting the control unit and the radiation detector does not include the cathode circuit of the phototube in this arrangement, the two units may be located at any distance from one another.

Work is now in progress on the design of a small phototube which will permit a considerable reduction in the size of the radiation detector. In addition, studies have been undertaken to develop a more sensitive fluorescent material for use in conjunction with the phototube. The developmental possibilities of an automatic exposing device similar to those employed in certain photographic cameras also are under investigation.

SUMMARY

An exposure meter for roentgenography is described. Complete details of the construction, calibration and operation of the instrument are given. A summary of fundamental photometric principles also is inincluded.

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LOW ABSORPTION ROENTGEN-RAY MEASUREMENTS FROM 10 TO 250 KILOVOLTS*

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THE inherent filtration of roentgen-ray tubes is by no means a new subject. The influence of the tube wall on the emitted radiation was discussed as early as 1912.[1] However, so long as the inherent filtration consisted entirely of a thin glass wall, the question of inherent filtration was not a serious one. With the advent of heavier glass walls and shockproof enclosures, the problem has become more complicated and of greater importance. The problem is of interest in the fields of both roentgen therapy and roentgenography. In roentgen therapy the inherent filtration of the tube unit influences both the intensity and quality of the beam—especially in the lower voltage range. In roentgenography the inherent filtration may determine the contrast of the roentgenogram, especially where low voltage techniques are concerned.

In actual practice there is no such thing as an unfiltered roentgen-ray beam. The thickness and composition of the glass wall of the tube may vary considerably in tubes designed for different purposes. In addition to the glass wall of the vacuum unit, the modern shockproof tube unit usually incorporates at least one, and in most cases two, other materials in the shockproof structure surrounding the tube and forming a part of the inherent filtration. In addition to glass, the other materials most often used are oil and bakelite.

As has been pointed out many times in the literature and in discussions, a statement of the quality of the roentgen beam expressed as the half-value layer at the voltage used would be adequate so far as comparing the results obtained from the various types of tube units is concerned. Without question, this should cover the problem. However, time does not seem to have dimmed the interest in nor to have reduced the discussion of inherent filtration and its equivalent in the most commonly used filter media: aluminum and copper.

Taylor and Singer[2] reported on the effect on the absorption curve for different kinds of glass of various thickness. A method is suggested for arriving at the inherent filtration. This study made use of a constant potential voltage source and covered a wide range of voltages.

Behnken and Nitka[3] made studies of the displacement of absorption curves by a change of inherent filtration. A tube with a Lindemann glass window was used. The practical use of the data presented is limited in that 31 kilovolts was the maximum voltage used.

A more recent report is that of Thoraeus.[4] In this study a special tube having an aluminum window 0.2 millimeter thick was used. The voltage source delivered constant potential and the work covered voltages from 50 to 100 kv. Equivalent inherent filtration from 0.2 to 10 mm. of aluminum was reported.

The purpose of this paper is to report a study of inherent filtration covering voltages from 10 to 250 kv. A tube having a lower absorption than any reported was used (approximately equivalent to 0.04 mm. of aluminum at 50 kv., peak), and the results given are reported for three kinds of filter material.

Since little information has been available regarding the change in intensity or quality of the roentgen beam by the various materials commonly included in shockproof roentgen-ray structures, a study has been made of this problem. Absorption data are shown for pyrex glass, oil, and bakelite at voltages from 10 to 250 kv.

* Read at the Forty-second Annual Meeting, American Roentgen Ray Society, Cincinnati, Ohio, Sept. 23–26, 1941.

ROENTGEN TUBE WITH BERYLLIUM WINDOW

In order to obtain in a tube wall the minimum absorption necessary for measurements of low initial filtration, it was deemed necessary to build a special tube, as nothing exactly fitting the purpose was available. A tube such as used by Thoraeus, with a 0.2 mm. aluminum window, would not cover all of the voltages at which it seemed desirable to work. Crystallographic roentgen tubes with Lindemann glass windows were available, but their voltage limitation would have made it impossible to cover any but the low voltage portion. Also, Lindemann glass is a chemical composition of lithium, beryllium, boron, and oxygen, which can vary in exact composition between windows, thus making it a very unsatisfactory medium to be always exactly reproduced or expressed.

FIG 1. Roentgen-ray tube with low absorption window of 1.3 mm. pure beryllium.

A special tube could have been constructed and operated on a pumping system, using a thin foil window of negligible absorption; however, it was felt that too great a difficulty would have been encountered in maintaining stable performance during the long hours of operation necessary to take the measurements and over the long period of time required to complete the study.

The ideal roentgen tube envelope material, from the viewpoint of minimum absorption, has long been recognized to be the metal beryllium (atomic number 4), but up until the present time it has never been made sufficiently dense, pure, or homogeneous to be vacuum-tight. It has been used commercially as a roentgen ray window, for a number of years, but merely as a window in a hooded anode type of tube where the surrounding glass envelope is the vacuum wall material. Of course, such a tube would in no way serve the purpose for minimum absorption roentgen-ray measurements.

A tube having a pure beryllium, vacuum-tight window was constructed and successfully used for all the measurements. As shown in Figure 1, the beryllium window 1.3 mm. in thickness was sealed on as an appendage to a standard, thick glass, 200 kv. therapy tube with 20° tungsten target anode of the circulating cooling liquid type.

The beryllium window, necessarily small in diameter, gave a 7.5 centimeter field of coverage at 50 cm. distance, which was more than adequate for thimble chamber measurements.

The space charge of the tube was extremely low and it was found possible to obtain 10 milliampere tube current at 10 kv. peak.

CIRCUIT AND SET-UP

A half-wave, single-phase, 60-cycle generator was used for all the measurements. It was selected because it is the most universal circuit in use, both for therapy and roentgenography. Also, the roentgen-ray output is the same except for small variation due to cables,[5,6] for both full-wave and half-wave single-phase rectifiers and when the roentgen tube is used self-rectified.

For voltages from 50 to 250 kv. peak, sphere gaps of a size consistent with standard practice were used to measure voltage. Below 50 kv. peak, a resistance voltmeter was used. The current was measured by a milliammeter in the high voltage circuit. A double diaphragm system was used, the first one at 9 cm. from the beryllium window, and the second at 16 cm.

At the lower voltages, possibly at less than 50 kv. peak, air absorption may alter the quality and quantity of radiation, as shown by Exner.[7] Therefore, for these voltages, precaution should be taken to use only measurements made at this same distance when comparisons are being made.

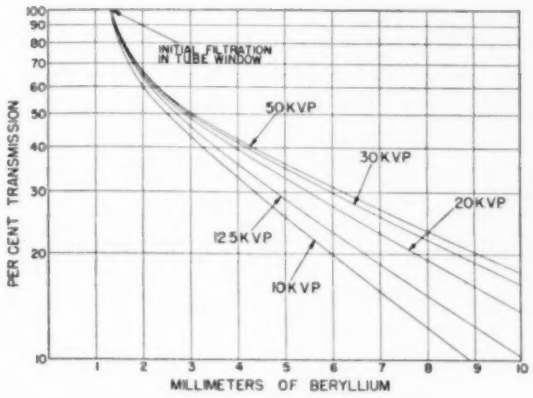

FIG. 2. Low voltage low initial absorption roentgen-ray transmission for beryllium.

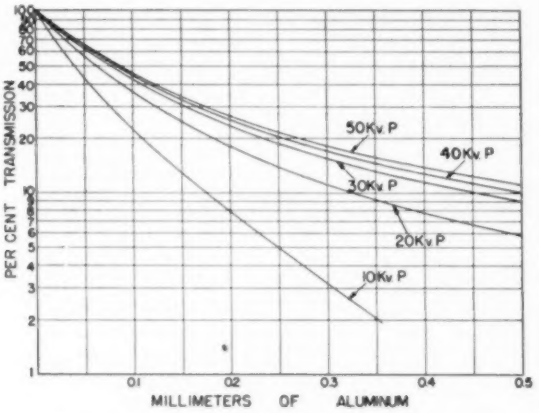

FIG. 3. Low voltage low initial absorption roentgen-ray transmission for aluminum.

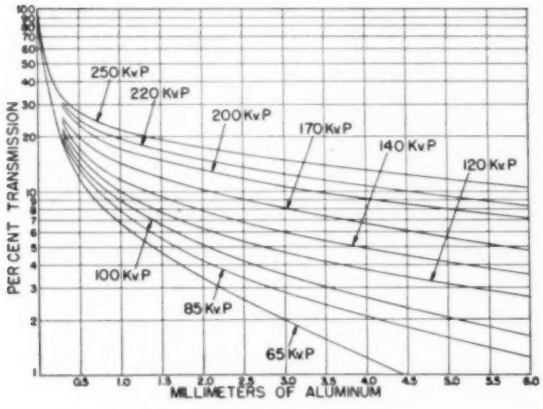

FIG. 4. Low initial absorption roentgen-ray transmission for aluminum.

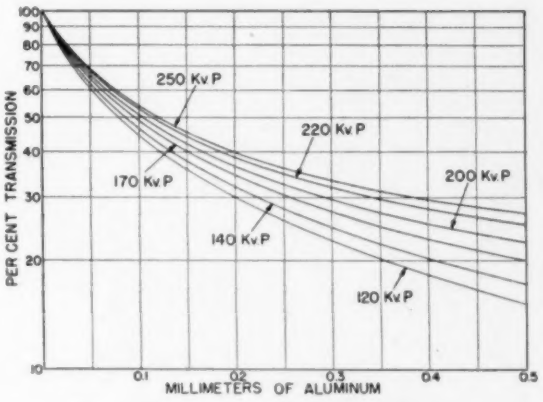

FIG. 5. Low initial absorption roentgen-ray transmission for aluminum.

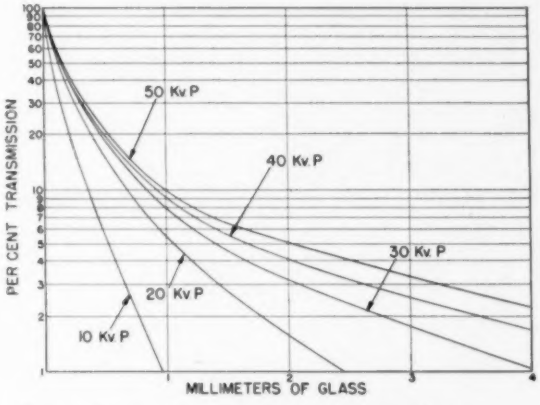

FIG. 6. Low voltage low initial absorption roentgen-ray transmission for Pyrex glass.

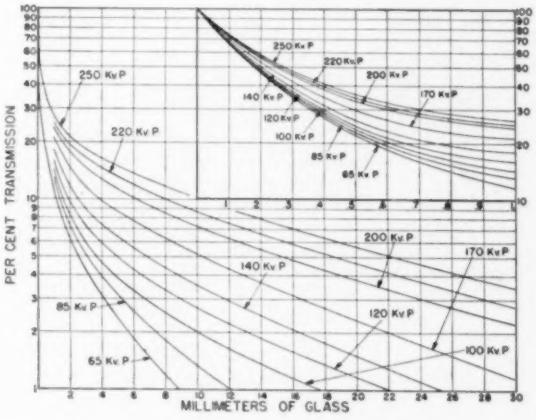

FIG. 7. Low initial absorption roentgen-ray transmission for Pyrex glass.

Also, it is apparent that at these lower voltages the same type of thimble chamber must be used.

ROENTGEN-RAY MEASUREMENTS

All measurements were made with a Victoreen thimble chamber in free air, at a

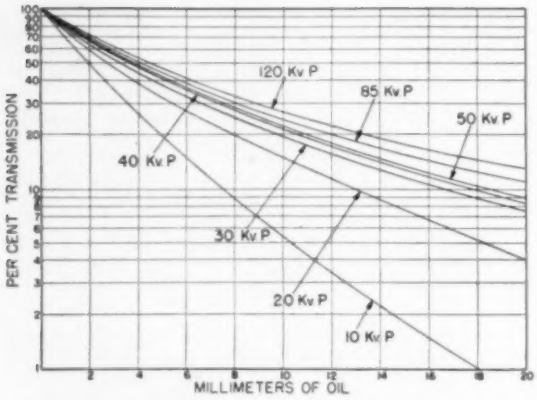

FIG. 8. Low voltage low initial absorption roentgen-ray transmission for transformer oil.

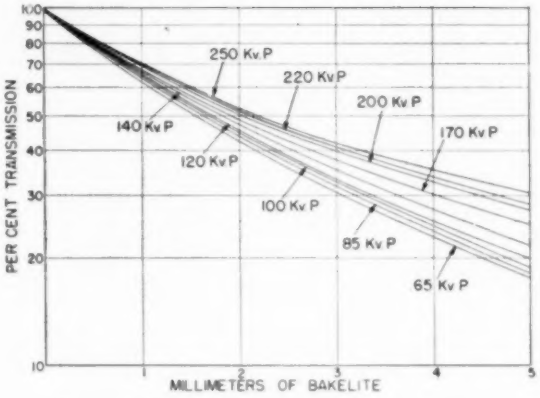

FIG. 11. Low initial absorption roentgen-ray transmission for natural bakelite.

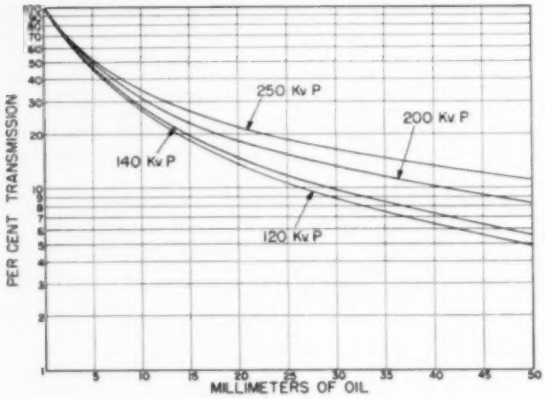

FIG. 9. Low initial absorption roentgen-ray transmission for transformer oil.

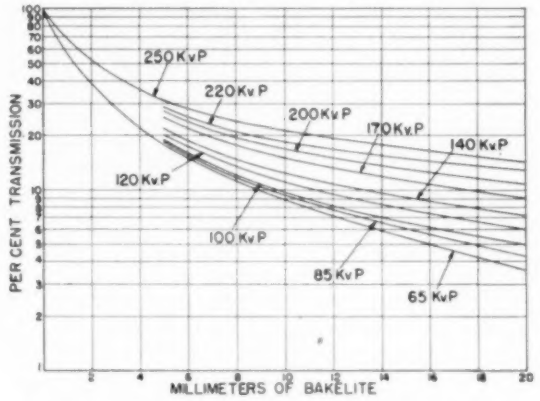

FIG. 12. Low initial absorption roentgen-ray transmission for natural bakelite.

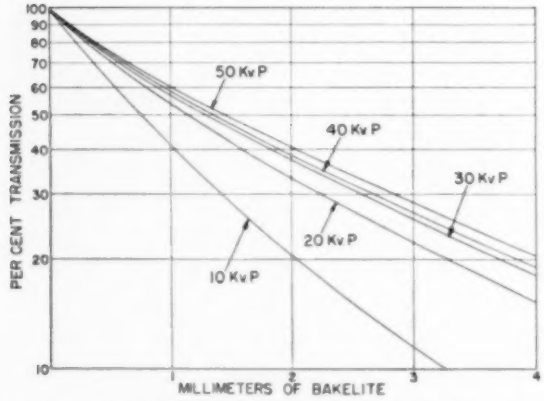

FIG. 10. Low voltage low initial absorption roentgen-ray transmission for natural bakelite.

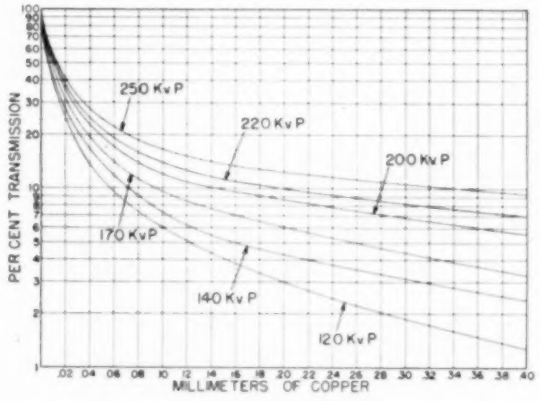

FIG. 13. Low initial absorption roentgen-ray transmission for copper.

distance of 50 cm., with 10 ma. tube current, for kilovoltages from 10 to 250 kv. peak. A number of thimble chambers with full scale ranges of 25, 100, and 250 r, were used with satisfactory reproduction of results. The resulting data seem to be consistent with other work on the problem when the differences in voltage sources, etc., are considered.

The work covered the greater part of a year, and it was always possible to reproduce any one or any series of readings

within the tolerance of experimental error.

Very early in the work there were indications that aluminum might not be the ideal material for absorption curves at the low voltages. It was hard to handle and preserve in the very thin sheets necessary, and the accuracy of the measurements was poor. The natural thing to do was to use the same material as used in the tube window—that is, beryllium. Our experience over a period of months in handling and using this metal has been very satisfactory. We wish to take this opportunity to advocate the

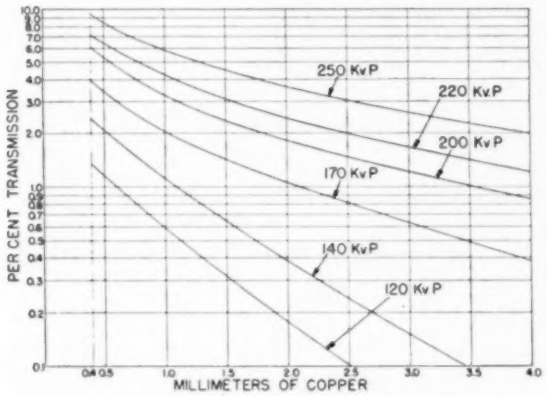

Fig. 14. Low initial absorption roentgen-ray transmission for copper.

use of beryllium as a medium in which to study and report absorption data taken at voltages up to 50 kv.

An attempt has been made to report and tabulate the results in a manner to make the information readily useful with a minimum of measurements and computation.

Determination of Inherent Filtration

The main use for the absorption curves presented here is, of course, to determine and express in the same medium, the inherent filtration of any combination of absorptive layers in a tube and housing. The data can be used in several different manners to obtain the inherent filtration.

One method that has been suggested is to measure the thickness of the glass, oil, and bakelite separately and sum up the resulting percentage transmission from the various curves. Unfortunately, this is not a very accurate method because the radiation is altered as it goes through each absorbing medium. However, for voltages of less than 100 kv. peak, and where the different layers are thin, a value accurate to a few per cent can be obtained without making any roentgen-ray measurements. This method can be used occasionally to advantage by the designer of roentgen tubes and housings to keep the inherent filtration in the proper range for the application being considered before constructing a model.

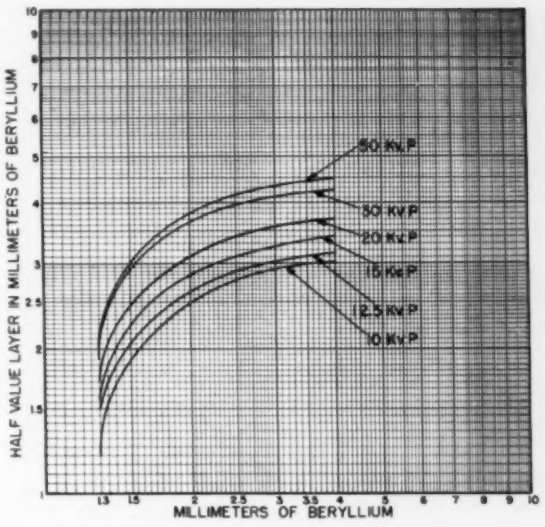

Fig. 15. Inherent filtration as a function of the half-value layer in beryllium.

Another method that has been used by

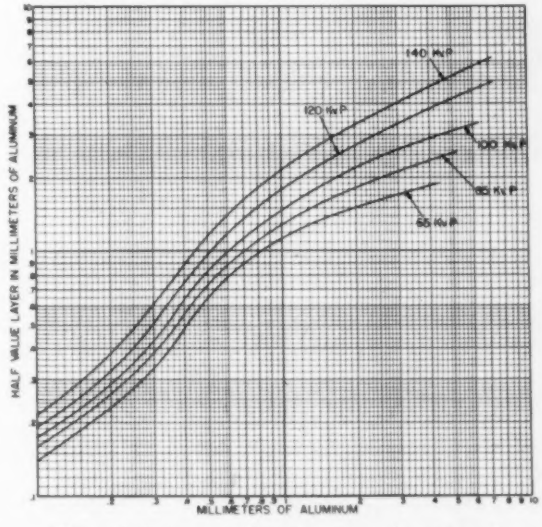

Fig. 16. Inherent filtration as a function of the half-value layer in aluminum.

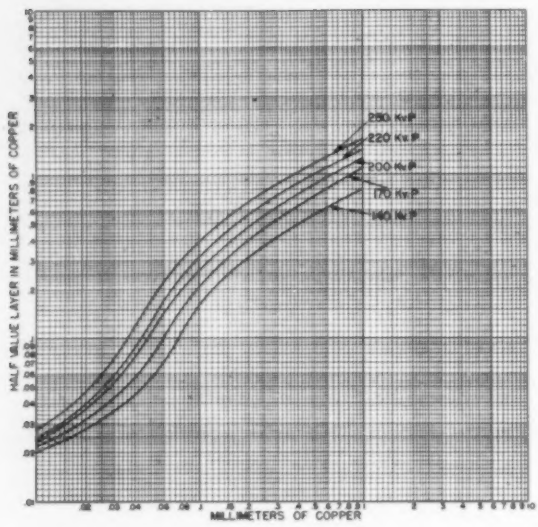

FIG. 17. Inherent filtration as a function of the half-value layer in copper.

Taylor and Singer[2] and is much more accurate than the above method is to graphically determine the point on the curve for the kilovoltage being used at which the same slope is obtained. That is, an absorption curve of roentgen-ray measurements is made at the desired kilovoltage using a filter material for which data are given. These values, plotted on a piece of transparent paper using the same scale of coordinates and matched to the same kilovoltage curve, will indicate the inherent filtration at the point coinciding with the zero added filtration value of the data taken. Three values of output are sufficient to establish the slope. It is very satisfactory to add 1 mm. and then 2 mm. of the filter material being used. Great care must be used in matching slopes so that, in general, this method could not be recommended for occasional usage.

A third method, as used by Thoraeus,[4] that can be recommended as the most satisfactory, makes use of the half-value layer. For simplicity in application, curves are given wherein the half-value layers obtained from the experimental data in Figures 2, 3, 4, 5, 13, and 14 are plotted against inherent filtration for beryllium, aluminum, and copper. In this method it is necessary to know only the half-value layer of the beam as taken directly from the roentgen-ray tube housing, without added filter. Figure 15 can be used to determine the inherent filtration for voltages of 50 kv. peak or less when the half-value layer has been determined in beryllium. For voltages from 65 to 140 kv. peak, the inherent filtration—when expressed in the half-value layer in aluminum—may be determined from Figure 16. Where the half-value layer is expressed in copper, Figure 17 may be used for voltages from 120 to 250 kv. peak.

SUMMARY

1. Curves showing the relation of the half-value layer in beryllium, aluminum, and copper to the inherent filtration at voltages from 10 to 250 kv. are presented.

2. Absorption data in beryllium, aluminum, copper, pyrex glass, oil, and bakelite, covering voltages from 10 to 250 kv., are shown.

3. Simple methods for the determination of the inherent filtration over a wide range of voltages are given.

4. The use of beryllium in making and reporting absorption data at very low voltages is recommended.

Work is under way for a later report covering voltages from 250 to 1,000 kv.

Recognition is due Mr. G. W. Hitchcock for having carried out the multitude of measurements necessary for this work.

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EDITORIALS

HISTOPLASMOSIS

IN 1906 Darling,[1] while searching for evidence of kala-azar in the smears from the spleen, liver, and bone marrow of all patients showing splenomegaly, who came to autopsy at the Ancon Hospital, discovered the organism known as the *Histoplasma capsulatum* of Darling.

This organism as found in the tissues is round or oval and measures from 1 to 4 micra in diameter. It has a deeply staining basophilic central mass or chromatin nucleus which is round, ovoid, or semilunar. This is surrounded by a clear space, which in turn is surrounded by a refractile achromatic rim. This is the yeast-like form of the fungus. Darling believed that it was a protozoan parasite.

Histoplasmosis or reticulo-endothelial cytomycosis is a generalized disease which varies widely in its clinical characteristics, and Meleney[2] stated that the disease may present one of the four following clinical pictures: (1) a systemic febrile disease similar to kala-azar, with enlarged liver and spleen, septic temperature, anemia, and leukopenia; (2) lymph node enlargement may predominate, simulating Hodgkin's disease, leukemia, lymphosarcoma, or aplastic anemia; (3) pulmonary symptoms may predominate. The infection is often complicated by, or superimposed upon, pulmonary or generalized tuberculosis; (4) the infection may begin as a small skin lesion which may sometimes develop into a generalized ulcerative skin condition.

Until the recent report by Key and Large[3] none of the cases presented any involvement of the joints or muscles, but these authors have recently had under their care a patient from whom sections of the specimen of the knee joint showed the typical *Histoplasma capsulatum* of Darling. The interest in their case lies in the fact that it is probably the first reported instance in which the disease is known to have involved a joint, and also because the case closely resembled tuberculosis of the knee joint and was so diagnosed until it was found that both guinea pig inoculation and tuberculin test were negative.

On clinical inspection the knee was considerably enlarged, being about half again as large as the other knee. The enlargement was symmetrical, the synovial cavity was distended, and the knee joint contained excess fluid; there was thickening of the periarticular tissues.

Roentgen studies of the knee showed thickening of the soft tissues and a moderate degree of bone atrophy with spotty atrophy of the bone structure. There was no decrease in the joint space and in neither the anteroposterior nor the lateral view was there evidence of new bone formation. Roentgen studies of the chest revealed somewhat atypical areas of consolidation rather well defined in the lower half of each lung field. It was not until the leg was amputated that the true diagnosis of the condition was evident in the pathologic findings.

The interest of the case, besides the unusual clinical manifestations, is that the patient was a native American and had never traveled outside this country, which indicates that the disease is not limited to the tropical or semitropical countries but may be found in the more temperate climates. Meleney believed that the disease is not as infrequent as might be supposed, that its infrequency is assumed because it is rarely diagnosed, that the organism is widely distributed throughout the world, and that it will undoubtedly be recognized

[1] Darling. Cited by Key and Large.
[2] Meleney. Cited by Key and Large.
[3] Key, J. A., and Large, A. M. Histoplasmosis of the knee. *J. Bone & Joint Surg.*, April, 1942, *24*, 281–290.

with increasing frequency in the future.

Therefore in a condition in which the changes about the knee joint give the roentgen picture of a tuberculous process, with atypical roentgen findings in the lung, the possibility of the disease being histoplasmosis should be considered.

With the great numbers of troops being sent into all parts of the world, particularly into the tropical zones, the increase in the incidence of histoplasmosis, as well as other protozoan diseases, may be expected.

OSTEOCHONDRITIS JUVENILIS OF THE ACETABULUM

INNUMERABLE articles have been written upon osteochondritis of the centers of ossification, particularly those involving the capital femoral epiphysis, as well as the epiphysis at the knee joint, but Lipscomb and Chatterton[1] recently pointed out that osteochondritis of the acetabulum has apparently only been called attention to by Froelich who described a case.

That the disease is more frequent than this single previously reported case would indicate is evidenced in this paper by Lipscomb and Chatterton in which they report three cases which have come under their observation. Therefore in some patients presenting themselves with an indefinite pain in the hip and associated limp, where the roentgen findings are apparently negative, the etiology may perhaps be found in an osteochondritis involving the acetabulum.

The growth of the acetabulum is a somewhat complicated mechanism and while the three primary centers of ossification which form the ilium, ischium, and pubic bone are present at birth, the greater part of the acetabulum is cartilaginous at birth. At about the tenth year of life the cartilage in the three bones at the acetabulum is reduced to a triradiate strip. Two or three secondary centers of ossification appear in this strip, one of which forms the os acetabulum at the age of twelve years.

From a study of the roentgenograms in their cases Lipscomb and Chatterton came to the conclusion that the disturbance seems to have been in the primary centers. Since the superior or weight-bearing surface of the acetabulum was chiefly involved that the iliac center of ossification at the acetabulum is the most likely to be affected.

The authors point out an observation which had been previously made by Jansen that variations in contour of the acetabulum may accompany osteochondritis of the capital femoral epiphysis. It is well known that structural variations of the acetabulum may occur in the Legg-Calvé-Perthes type of osteochondritis and that regions of condensation and rarefaction similar in appearance to that of the femoral neck adjacent to the epiphyseal cartilage are present in the subchondral portion of the bone.

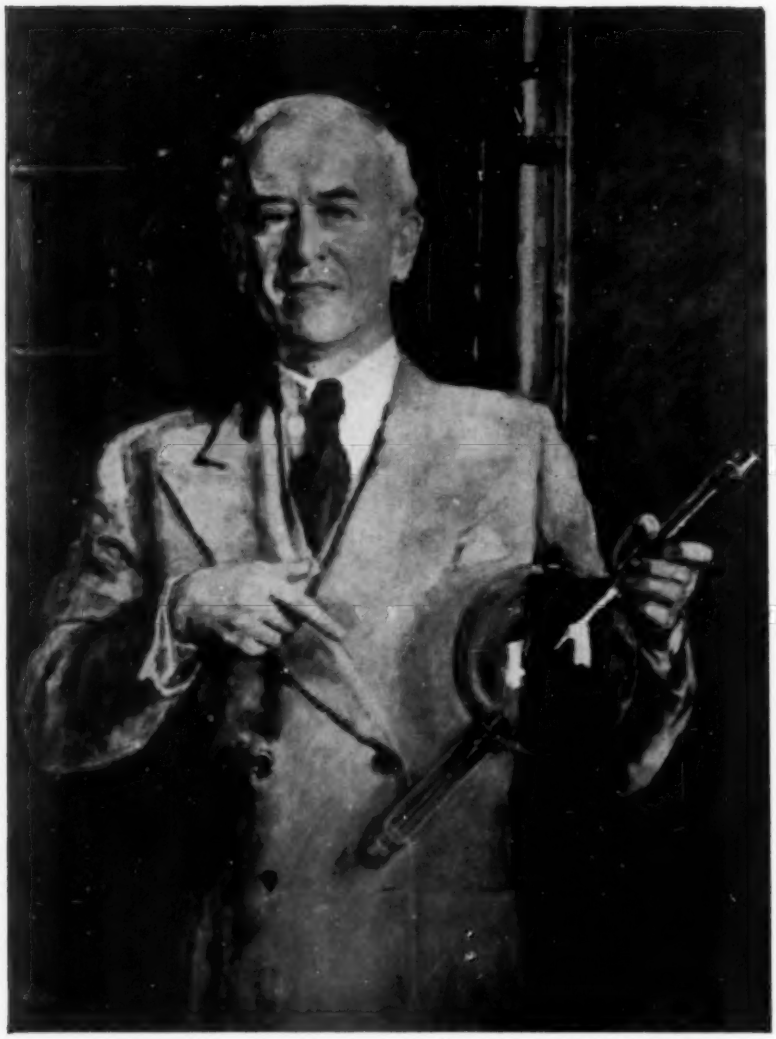
Lipscomb and Chatterton believe that in a certain percentage of cases structural changes similar to those in the femur take place in the acetabulum coincidentally with, and not secondarily to, the changes in the femoral head and neck, and that in certain cases there may be structural change in the acetabulum without any detectable change in the head and neck of the femur, the changes being primary in the acetabulum.

In the cases which they reported the symptoms were unilateral, yet in practically all instances both acetabula presented roentgenographic evidences of changes of varying degree.

Because the structural changes seem to involve the superior or weight-bearing surface of the acetabulum the authors are of the opinion that the changes are probably due to a circulatory disturbance which is perhaps aggravated by trauma.

The changes are most likely to occur at the superior or weight-bearing surface of the acetabulum and in suspected cases this portion of the acetabulum should be examined with great care, keeping in mind the possibility that any changes seen may be slight structural variations from the normal and not those due to a disease process.

[1] Lipscomb, P. R., and Chatterton, C. C. Osteochondritis juvenilis of the acetabulum. *J. Bone & Joint Surg.*, April, 1942, *24*, 372–381.



From the portrait by Nikol Schattenstein

I. SETH HIRSCH
1880–1942

"HE WISHES to knit anew his connections with his oldest friends, to continue those recently formed, and to win other friends among the rising generation for the remaining course of his life. He wishes to spare the young those circuitous paths, on which he himself had lost his way." Thus wrote Goethe in his "Introduction to the Propyläen," symbolic of the gateway of the Acropolis of Athens, and this quotation served Smith Ely Jelliffe as an appropriate introduction to the Periclesian work of Dr. I. Seth Hirsch in the Appreciation prefacing the text entitled "Contributions to Radiology."

I. Seth Hirsch was born in New York City on December 3, 1880. He attended the College of the City of New York from 1895 to 1898, and received the degree of Doctor of Medicine from the College of Physicians and Surgeons of Columbia University in 1902. In 1920 he received a Diploma in Radiology from Cambridge University, England.

Curiosity drove him to the microscope and the bacteriological laboratory, and this searching went further, into the new science opened up by the discoveries of Roentgen. In 1910, after a Civil Service examination, he was appointed Director of Roentgenology at Bellevue Hospital. Here his love for order caused him to re-arrange the entire organization and build a radiological laboratory of note, and for sixteen years endless opportunities for study and service made his life an arduous, happy, and busy one. When Bellevue Hospital was incorporated with other New York City hospitals into the group known as Bellevue and Allied Hospitals, which included Harlem, Fordham and Gouveneur Hospitals, Dr. Hirsch became the General Director, a post which he held until 1926.

From 1913 to 1917 he was Director of Roentgen Laboratories of the New York Post Graduate Hospital, and for the three years before the entry of the United States into the first World War he was Professor of Roentgenography at the Medical College of this institution.

Entering the Medical Corps of the United States Army as a Captain in 1917, Dr. Hirsch was subsequently promoted to the rank of Major.

In 1920 his "Principles and Practice of Roentgen Technic" appeared, to be followed in 1925 by his "Principles and Practice of Roentgen Therapy," a pioneer work in English deemed worthy of translation by Holzknecht and Spiegler. Two books that he had in preparation at the time of his death will be published posthumously. Dr. Hirsch also served as an Associate Editor and Director of the Department of Technique and New Devices of the *American Journal of Electrology and Radiology*.

He was Director of the Roentgen-Ray Department of Beth Israel Hospital in New York City from its inception until 1917, and again from 1929 until his death, where with the aid of Myron M. Schwarzschild, physicist, he was able to delve into the many physical intricacies that surrounded the growing field of radiology. He was Vice President of the Radiological Society of North America in 1927 and 1928, and a Fellow of the American College of Physicians.

A volume entitled "Contributions to Radiology," consisting of forty-five scientific articles, was published in his honor in 1939 by a group of former pupils, colleagues and friends. These papers originally appeared in *Radiology*. The Editorial Committee in charge of compiling this volume called it "a literary memorial to testify to the admiration, respect and affection in which Radiologists hold I. Seth Hirsch and a recognition of his services as investigator and teacher." At a dinner to commemorate the publication of this volume Dr. Hirsch was presented with an oil painting of himself executed by Nikol Schattenstein. The portrait first hung in the New York University College of Medicine and, more recently, in his home. He was the author of a play called "The Unlistening Street," a scene from which was dramatized by a group of well known actors on the night of the dinner.

At the time of his death Dr. Hirsch was serving as Consultant to the New York State Compensation Commission. Since 1933 he had been Professor of Radiology at the New York University College of Medicine.

Throughout the years there have come from his work studies characterized by originality, freshness of viewpoint, and a striving for simplification. Particularly noteworthy, however, are his most recent ingenious contributions to the analysis of cardiodynamic phenomena by permanent roentgen records. He also opened up a new field of kymophonoroentgenography, thus making possible the studying and recording of sound phenomena of the heart and the valvular actions responsible for them. He has "hitched" the heart to the roentgen machine in such a manner as to picture this organ in definite phases of its activity.

He was a sculptor of talent and to his clinical research work brought a highly cultivated appreciation of the beauties of

nature derived from his sense of beauty of line and its organization. Horticulture was another of his avocations.

Broadly speaking, the fundamental significance of I. Seth Hirsch lay in the application of his scientific genius to a new and growing specialty. It is upon his contributions and work in the field of radiology, especially his ability to organize and teach, that his permanent fame securely rests. He had a genius for leadership, was steadfast in purpose, tenacious of principles in which he believed, patiently persevering, and fearless, constructive and clear in vision, with a sure sense of the immediately obtainable. His practical sagacity, his sense of reserve strength, combined with wide knowledge, experience and prestige which came to him with the passing years, gave I. Seth Hirsch a unique position in the realm of radiology.

Dr. Hirsch died on March 24, 1942, after a short illness. He leaves a widow, the former Miss Lila Calhoun Hindsman, of Fort Worth, Texas.

M. H. POPPEL

WALTER COX BARKER
1878–1941

WALTER COX BARKER died at his home in Philadelphia on December 26, 1941 after a short illness. Dr. Barker was born in 1878. He received his preliminary education at the Central Manual Training School and the Brown Preparatory School. A few years after his graduation from the Hahnemann Medical College of Philadelphia he became interested in roentgenology and began its sole practice in 1908. Besides carrying on his private practice he was for many years Radiologist to the Broad Street Hospital and Consulting Radiologist to the Hahnemann Hospital. He was a past president of the Philadelphia Roengen Ray Society, a member of the American Roentgen Ray Society and of the Radiological Society of North

America and a diplomate of the American Board of Radiology.

Possessed of a kindly and courteous disposition, a keen knowledge of his chosen speciality, and sound diagnostic judgment, he was held in high esteem. In addition to his membership in the Philadelphia County Medical Society he was a member of the Pennsylvania State Homeopathic Medical Society and the American Institute of Homeopathy. In the programs of the latter he was often a participant. In his passing, the profession has lost an able and unassuming member, and his colleagues, a staunch and loyal friend.

RALPH S. BROMER

SIR WILLIAM HENRY BRAGG
1862–1942

THE life and work of the great physicist, Sir William Henry Bragg, are of interest to all radiologists. The following obituary is reprinted from the *New York Times* for March 13, 1942:

Sir William Henry Bragg, who shared the Nobel Prize with his son, William Lawrence Bragg, in 1915 for their work on X-rays and crystals, died today [March 12] at the age of 79. Also in 1915 the father and son received the Barnard Medal of Columbia University for the same type of research.

Though academic honors were heaped on Sir William through the years, he probably was best known for his work in popularizing science. His putting of abstruse matters in simple language caused him to be called the "children's scientist."

Sir William, the famous investigator of the architecture of atoms and molecules as shown by the way they disturb and scatter X-rays passing through them, shared with his son in the development of the X-ray spectrometer which aided in elucidating the arrangements of atoms and crystals.

In 1924 the British physicist summed up the knowledge which the X-ray had furnished concerning the versatile carbon atom, the basis of every plant and animal that lives. Declaring that carbon "is the commonest atom," he held that "oxygen bulks largest, and half the earth, so far as we can observe, is made of oxygen."

On a visit here in 1930 Sir William read extracts from Faraday's diary, which never before had been published, before an audience of 400 physicists and chemists at Cornell University. It was also during this visit to America that 200 scientists attended a luncheon at the Hotel Astor in his honor, when an honorary membership in the New York Meteorological Society was presented to him.

Sir William took time from his researches and wrote a book for the general reader which he called "The Universe of Light." It was published here in 1933. In another work, "Concerning the Nature of Things," Sir William devoted a substantial part to the nature of crystals.

When Sir William made his first address as president of the British Association for the Advancement of Science in Glasgow in 1928 he flatly told his audience that man has a soul. The distinguished scientist shook his finger at his 3,000 listeners and declared:

"Science is not setting forth to destroy the soul, but to keep body and soul together."

Born on July 2, 1862, at Wigton, Cumberland, Sir William attended King William's College, Isle of Man, and Trinity College, Cambridge. He became Professor of Mathematics and Physics at Adelaide, South Australia, in 1886, Cavendish Professor at Leeds in 1909 and Quain Professor of Physics in the University of London in 1915.

During the first World War Sir William acted in an advisory capacity to the British Admiralty, especially on the problem of submarine detection. He was created K. B. E. in 1920 and in the same year was elected honorary fellow of Trinity College, Cambridge. He was president of the Physical Society of London and in 1923 was appointed Fullerian Professor of Chemistry at the Royal Institution and director of the Davy-Faraday research laboratory. He succeeded Sir Arthur Keith as president of the British Association for the Advancement of Science in 1928.

Since the outbreak of the present conflict Sir William had served as chairman of an especially appointed scientific food committee.

His wife, Gwendoline, whom he married in 1889, was the daughter of the late Sir Charles Todd. She died in 1929.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual Meeting: Atlantic City, N. J., Oct. 6–9, 1942.

American College of Radiology
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual meeting: Atlantic City, N. J., June 10, 1942.

Section on Radiology, American Medical Association
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Atlantic City, N. J., June 8–12, 1942.

Arkansas Radiological Society
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Arkansas. Meets every three months and also at time and place of State Medical Association.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Drake Hotel, Chicago, Ill., Nov. 30-Dec. 4, 1942.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Section on Radiology, California Medical Association
Secretary, Dr. J. D. Coate, 434 Thirtieth St., Oakland, Calif.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

Radiological Section, Los Angeles County Medical Association
Secretary, Dr. Donald R. Laing, 65 North Madison Ave., Pasadena, Calif. Meets on second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N. Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Chicago Roentgen Society
Secretary, Dr. Warren W. Furey, 6844 S. Oglesby Ave., Chicago, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, J. O. Newton, 13921 Terrace Rd., East Cleveland. Meets at 6:30 P.M. at Midday Club on fourth Monday each month, October to April, inclusive.

Denver Radiological Club
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida State Radiological Society
Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Florida. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. R. C. Pendergrass, Prather Clinic Bldg., Americus, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Greater Kansas City
Secretary, Dr. P. E. Hiebert, 811 Huron Bldg., Kansas City, Kansas. Meets third Thursday of each month at a place designated by the president.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in April.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society meeting.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. J. P. Medelman, 572 Lowry Medical Arts Bldg., St. Paul.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. Hugh F. Hare, Lahey, Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

Radiological Society of New Jersey
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

New York Roentgen Society
Secretary, Dr. Paul C. Swenson, Presbyterian Hospital, New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Ohio Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport. Next meeting, Necho Allen Hotel, Pottsville, Pa., May 15–16, 1942.

Philadelphia Roentgen Ray Society
Secretary, Dr. B. R. Young, Temple University Hospital. Meeting first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. H. W. Jacox, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho, Annual meeting in Denver, summer of 1942.

St. Louis Society of Radiologists
Secretary, Dr. W. K. Mueller, University Club Bldg. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Francisco Radiological Society
Secretary, Dr. H. A. Hill, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months at Toland Hall, second at Lane Hall.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. L. W. Baird, Scott and White Clinic, Temple, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. C. H. Peterson, 603 Medical Arts Bldg., Roanoke, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. K. J. Holtz, American Bank Bldg., Seattle. Meets fourth Monday each month, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets monthly on third Thursday from November to June inclusive, at 8:15 P.M., 32 Welbeck St., London.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress.
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N.S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevskv. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

AMERICAN RADIUM SOCIETY

The Twenty-seventh Annual Meeting of the American Radium Society will be held in Atlantic City, New Jersey, in the Viking Room of Haddon Hall, on Monday and Tuesday, June 8 and 9, 1942. The following program has been arranged:

PROGRAM

Monday Morning, June 8, 1942

Presidential Address. Hayes E. Martin, M.D.

Pulmonary Metastases and Pneumonitis Following Radiation Therapy from Cancer of the Breast. Eugene P. Pendergrass, M.D., and George White, M.D., Philadelphia.

Frequency, Clinical Course, and Management of Metastases from Cancer of the Breast. Jacob R. Freid, M.D., and Henry Goldberg, M.D. (by invitation), New York.

Fractures of the Rib Cage Following Interstitial Radium Therapy for Cancer of the Breast. Hoke Wammock, M.D. (by invitation), Philadelphia.

Radiation Osteitis and Fractures Following Irradiation with Report of Five Cases of Fractured Clavicle. Danely P. Slaughter, M.D. (by invitation), New York.

The Effect of Roentgen Therapy on the Heart: A Clinical Study. John E. Leach, M.D. (by invitation), New York.

Monday Afternoon, June 8, 1942

System of Tumor Dosage, Records, and Technique as Employed at the Brooklyn Cancer Institute. William E. Howes, M.D., Brooklyn, New York.

Radiation Therapy in Cancer of the Esophagus: An Analysis of 83 Cases Observed During the Last Decade. Ernst A. Pohle, M.D., Ph.D., and Roland R. Benson, M.D. (by invitation), Madison, Wisconsin.

Million Volt Therapy for Intrathoracic Neoplasms. William L. Watson, M.D., and Jerome A. Urban, M.D. (by invitation), New York.

Janeway Lecture: The Rôle of the Gynecologist in the Field of Cancer. William P. Healy, M.D., New York.

Monday Evening June 8, 1942

ANNUAL BANQUET

Benjamin West Room, Haddon Hall

Tuesday Morning, June 9, 1942

Cancer of the Uterus: The Results of Present Method of Radium Therapy as Influenced by Stage and Grade of the Lesion. H. H. Bowing, M.D., and R. E. Fricke, M.D., Rochester, Minnesota.

The Elimination of Radiation Injuries in the Treatment of Carcinoma of the Cervix. Charles L. Martin, M.D., Dallas, Texas.

Introducer for Inserting Multiple Capsules of Radium in the Uterus for the Treatment of Corpus Cancer. James F. Nolan, M.D. (by invitation), and A. N. Arneson, M. D., St. Louis, Missouri.

Treatment of Rhinoscleroma. Guillermo Montana, M.D., and Luis Vargas, M.D., Mexico (by invitation).

The Histological Changes Produced by Fractionated Irradiation of Squamous Cell Epithelioma of the Mouth and Throat. Milton Friedman, M.D., and John W. Hall, M.D. (by invitation), New York.

Surgery and Radium Therapy in Cancer of the Lower Lip. Isaac Levin, M.D., New York.

Facial and Oral Prostheses (a Motion Picture). Andrew Ackerman, D.D.S., New York (by invitation).

Tuesday Afternoon, June 9, 1942

The Effect of Increasing the Dose of Roentgen Therapy to the Parametria on the Rate of Cure. William P. Healy, M.D., and Gray H. Twombly, M.D. (by invitation), New York.

New Method of Applying Radium in Uterine Conditions. Ira I. Kaplan, M.D., New York.

Radon Treatment of Cancer of the Cervix Uteri (a Motion Picture in Technicolor). Frank E. Simpson, M.D., J. Ernest Breed, M.D., and James S. Thompson, M.D. (by invitation), Chicago.

A Remote Controlled Radon Plant and Automatic Measuring Apparatus. John E. Rose, Baltimore, Maryland (by invitation).

Protection of Radium under War Conditions. L. F. Curtiss, Washington, D.C. (by invitation).

The Centrosomes in Some Neoplastic Growths. Gonzales Guzman, M.D., Mexico City (by invitation).

Further Studies of Deep Tissue Reactions Following Irradiation for Cancer of the Upper Respiratory Tract. Howard H. Ashbury, M.D., Baltimore, Maryland.

MINNESOTA RADIOLOGICAL SOCIETY

The Spring Meeting of the Minnesota Radiological Society was held at the Mayo Foundation House, Rochester, Minnesota on Saturday, March 28, 1942. The following program was given in the afternoon, after luncheon at the Mayo Foundation House, by members of the staff of the Mayo Clinic:

Roentgenologic and Pathologic Studies of Prepyloric Ulcer. W. C. MacCarty, Jr., M.D., and B. R. Kirklin, M.D.

Cholelithiasis in the Functioning Gallbladder. Richard A. Twyman, M.D., and B. R. Kirklin, M.D.

Histopathology of the Protruded Intervertebral Disc. J. McK. Ivie, M.D., and J. W. Kernohan, M.D.

Infections of the Intervertebral Disc. Ralph K. Ghormley, M. D.

Roentgenologic Aspects of Pseudomyxoma Peritonei. David G. Pugh, M.D.

Primary Tumors of the Ribs. Frank J. Rigos, M.D., and John D. Camp, M.D.

Roentgen Treatment of Erysipelas. James D. Sheldon, M.D., and W. D. Popp, M.D.

The Radiologist in the U. S. Navy. Gordon Perisho, Lieutenant, M.C., U. S. N.

Platybasia. Robert D. Moreton, M. D., and John D. Camp, M.D.

Prognosis in Carcinoma of the Stomach in Relation to Duke's Type and Broders' Grade of Malignancy. G. R. Dochat, M.D., and M. D. Dockerty, M.D.

Dysgerminomas with Reference to Radiosensitivity. Robert D. Moreton, M.D., and E. T. Leddy, M.D.

Some Interesting Case Reports. David G. Pugh, M.D., and A. U. Desjardins, M.D.

The evening program consisted of a fellowship hour, dinner, and business meeting, after which Dr. Patrick Wu, of Peiping, China, gave an address entitled "China Speaks."

CHICAGO ROENTGEN SOCIETY

At the meeting of the Chicago Roentgen Society held on April 9, 1942, the following officers were elected for the coming year: *President*, Dr. Chester J. Challenger; *Vice President*, Dr. Earl E. Barth; *Secretary*, Dr. Warren W. Furey.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers as space permits.

RHEUMATIC FEVER. STUDIES OF THE EPIDEMIOLOGY, MANIFESTATIONS, DIAGNOSIS, AND TREATMENT OF THE DISEASE DURING THE FIRST THREE DECADES. By May G. Wilson, M.D., The New York Hospital and Department of Pediatrics, Cornell University Medical College, New York City. Cloth. Price, $4.50. Pp. 595, with 47 illustrations and 79 figures. The Commonwealth Fund; London: Oxford University Press, 1940.

If anyone is inclined to doubt the supreme importance of acute rheumatic fever as a cause of mortality and morbidity in the human race, let him ponder over the following statements. Cardiac disease is the leading cause of death in the United States. Sixty to 80 per cent of heart disease, in persons under forty years of age, is of rheumatic origin. In New York City, rheumatic heart disease results in more deaths in persons under twenty years of age than does pulmonary tuberculosis and all of the following disorders taken together,—pertussis, meningococcus meningitis, measles, diphtheria, scarlet fever and poliomyelitis. When one considers the amount of funds available for necessary research in the latter disease, it appears as though this much more important condition, which also affects young persons primarily, has been slighted grievously. As a public health problem, the condition now ranks with tuberculosis and syphilis. All of which leads the reviewer to state what he has long thought, namely, that if one should devote the major portion of his entire life to an intensive study of the prevention or cure of rheumatic fever, and would discover a single small fact which would eventually lead at least to some means whereby the heart might be spared, he would have made a worthy contribution to humanity, of inestimable value.

The monograph of Dr. May G. Wilson, which has been sponsored by the Commonwealth Fund, presents the results of more than twenty years' study of the etiology, epidemiology, manifestations, course, diagnosis, prognosis, and treatment of childhood rheumatic fever. Her work, which is based chiefly on her long experience in the cardiac clinics and wards of the New York Nursery and Child's Hospital, is probably the most authentic and complete summing up of recent data pertinent to rheumatic fever which has appeared in the medical literature. It should engage the careful attention of all physicians who encounter patients with the malady or those suffering from its sequelae. There is a most complete bibliography following each one of the five subdivisions of the work which should be of great assistance to all who are interested in the literature dealing with any aspect of the disease.

Many fascinating phases of the disease have been attractively presented and all could be appropriately reviewed if space were available. Only a few, however, can be discussed.

The etiology of rheumatic fever is unknown. The author considers that it is probably an infection of a low degree of contagiousness but the causative agent has not been isolated. Nor has it been possible to reproduce the disease in animals. The earlier concept of a streptococcemia, which is no longer tenable, has been superseded by the allergic hypothesis which is based largely on speculative grounds. Of greatest importance is the observation, supported by genetic analysis, that hereditary susceptibility underlies the familial incidence of the disease, although it probably is not the sole condition essential for its development. If a case occurs in any given family, some prediction of the sibling incidence may be made. For example, if one child is rheumatic, it might be expected that the chances of susceptibility among subsequent siblings would be one to four or one to two, or more, depending on the genetic background. In this study, it was concluded that the hereditary mechanism was probably a single autosomal recessive gene.

In studying the course of the disease, Dr. Wilson has had available the records of 717 patients. In 348 the course of the condition has been known for ten years, in 199 for fifteen years, and in 148 for twenty years. It was found that the initial attack occurred in one-fifth when under five years, one-half at the ages of

five through eight years, and one-fourth over eight years. It is generally agreed that the initial attack of rheumatic fever does not often occur after the age of fifteen years. Growing and joint pains, chorea, and polyarthritis each occurred as sole manifestations at the onset in one-fourth of the cases. In only 3 per cent was active carditis the only evidence of the disease at the beginning; but either alone, or with polyarthritis or chorea, it was the earliest indication of the condition in 15 per cent. Eighty-five per cent experienced one or more recurrences. It should be noted, however, that after the age of puberty, regardless of the age of onset, there is a diminished tendency toward exacerbations of rheumatic activity.

While cardiac disease has been known to accompany or follow acute rheumatic fever commonly, it is unlikely that physicians appreciate the remarkably high incidence of this complication. For example, it was found that in 325 patients who were under observation either before or within one year after the onset, cardiac involvement occurred in 85 per cent. *This observation is convincing evidence that the heart is probably always involved at the onset of rheumatic fever.* With such startling figures one naturally would inquire as to the criteria employed which were taken to indicate cardiac involvement. The heart was said to have been affected when characteristic murmurs were noted, or if it could be demonstrated that there was enlargement either with or without associated significant evidence of valvular disease. It is emphasized by the author that the most important single early diagnostic criterion is cardiac hypertrophy. This is readily recognized by physical and roentgenographic examination when pronounced, but the usual roentgenologic criteria offer little assistance in determining the most important finding of early cardiac enlargement. The method which was finally utilized by the author was radioscopy, which included teleradioscopy and fluoroscopy. Absolute reliance should not be placed on measurement of the frontal plane of the cardiac silhouette. A discussion of her findings is given with comments on the reliability of methods of estimating the size of the heart which should have a special appeal to all who have an interest in that subject.

Additional divisions are devoted to prognosis, to an appraisal of methods of determining activity and extension of the disease such as determination of the vital capacity, multiple electrocardiographic studies, the exercise test, the leukocyte count, the sedimentation test, and the presence or absence of fever. The concluding chapters deal adequately with "specific" and symptomatic treatment.

In conclusion, let it be said that this publication is not just another monograph dealing with an important subject, but one based upon a well planned study, guided by an observer of ability who apparently has an ardent interest in the field, and one which is a definite contribution to medicine. The Commonwealth Fund is to be congratulated in sponsoring such a valuable, carefully executed and long-continued study.

Cyrus C. Sturgis

The Modern Treatment of Syphilis. Second edition. By Joseph Earle Moore, M.D., Associate Professor of Medicine, The Johns Hopkins University; Physician-in-charge Syphilis Division of the Medical Clinic and Visiting Physician, The Johns Hopkins Hospital, Baltimore; Special Consultant, United States Public Health Service. With the Collaboration of Jarold E. Kemp, M.D., Associate in Venereal Diseases, Johns Hopkins University; Harry Eagle, M.D., Past Assistant Surgeon, United States Public Health Service and Lecturer in Medicine, Johns Hopkins University; Paul Padget, M.D., Associate in Medicine, Johns Hopkins University; and Mary Stewart Goodwin, M.D., Instructor in Pediatrics, Johns Hopkins University. Cloth. Price, $7.00. Pp. 674, with 92 illustrations. Springfield, Illinois: Charles C Thomas, 1941.

The first edition of "The Modern Treatment of Syphilis," published in 1933, has been revised by Moore and his collaborators and many new major items have been introduced into the contents of this second edition which have made it thoroughly modern.

This book fills a need in medicine as it compresses, into one handy volume, a mass of information which deals authoritatively and comprehensively with the subject. The senior author has had for over a quarter of a century an extensive experience with the disease in the syphilis division of The Johns Hopkins Hospital, in private practice, and in the World War of 1917. This, when associated with his familiarity with the literature, his thorough knowledge

of internal medicine, and his ability to express his thoughts in a lucid and concise manner, makes this publication the most satisfactory one of its type which is available.

Certain important points, in addition to being discussed fully, are emphasized in an abbreviated manner at intervals throughout the book. For example, on page 229 are ten principles, expressed so clearly that no one could possibly misunderstand them, relating to the early treatment of syphilis. This may seem somewhat obvious to some but simplicity, conciseness, clearness and emphasis are qualities of writing most desired by the medical student and practitioner, and in fact any member of the medical profession who is reading in a field in which he is not an expert.

Various ingenious devices are utilized to impress important facts. For example, in the section on the treatment of latent syphilis is a diagram which stresses the value of treatment of latent syphilis. In this it is shown that death from antisyphilitic treatment occurs only once in two thousand times, whereas in patients with untreated syphilis the chance of death from the disease is one in four. This fact must be somewhat disconcerting to the physician who exhibits the case of the otherwise healthy untreated elderly syphilitic as evidence that patients may remain in good health despite the lack of therapy. Of course, such cases are known to all of us but the chances of its occurrence have not been emphasized previously in such a convincing manner.

The chapter on the management of cardiovascular syphilis is written, as it should be, from the standpoint of the internist. An excellent discussion is given of the correlation between the clinical and the pathological features of the condition, and comments are made on the early diagnosis of this complication. There are two points which have received special emphasis: first, that with the exception of neurosyphilis, and perhaps even exceeding this, cardiovascular involvement is the most frequent cause of death in acquired syphilis, and second, to cope successfully with it, necessitates the recognition of the condition when it is in the uncomplicated aortitis stage. To accomplish this, the possibility of its occurrence should always be kept in mind in all patients who have had syphilis for ten years or longer. Of the various diagnostic criteria available, the most important single one is the roentgenological demonstration of dilatation of the first portion of the aorta.

In the specific treatment of cardiovascular syphilis, it is advised that arsphenamine (606) be completely avoided, even in minimal amounts, and that mapharsen, neoarsphenamine or bimarsen be employed in small doses. In order to avoid the Herxheimer reaction (therapeutic shock) it is recommended that treatment be initiated and continued with bismuth and iodides for a period of eight to twelve weeks, and finally, that continuous therapy should be given for a minimum interval of two years.

And, finally, the results and a full discussion are given of six systems of treatment which have been used at Johns Hopkins Hospital for neurosyphilis. Surely advice concerning this perplexing and controversial problem will be welcome from one who has had such an extensive experience.

Certainly the reviewer can recommend wholeheartedly this compact volume on syphilis to all medical students and practitioners, and all others interested in the disease, without the slightest fear that any of them will be disappointed in the presentation of any phase of the disease.

Cyrus C. Sturgis

Surgical Diseases of the Spinal Cord, Membranes, and Nerve Roots: Symptoms, Diagnosis, and Treatment. By Charles A. Elsberg, M.D., Emeritus Professor of Neurological Surgery, College of Physicians and Surgeons, Columbia University; Emeritus Director of Neurosurgery, Neurological Institute of New York; Consulting Surgeon, Mount Sinai Hospital, New York City. With Chapters by Cornelius G. Dyke, M.D., Associate Professor of Radiology, College of Physicians and Surgeons, Columbia University; Director of the Department of Roentgenology, Neurological Institute of New York, New York City, and Abner Wolf, M.D., Assistant Professor of Neuropathology, College of Physicians and Surgeons, Columbia University, Attending Pathologist, Neurological Institute of New York, New York City. Cloth. Price, $14.00. Pp. 598, with 249 illustrations. New York: Paul B. Hoeber, Inc., 1941.

In the Preface the author states that this volume has been prepared to state concisely "the essential features of diseases and injuries

of the spinal cord, membranes, and nerve roots, which the surgeon may encounter, as well as to give the indications for operative intervention and to describe the technique of operations upon the spinal cord and nerve roots."

The reviewer, as a radiologist, therefore hesitates to comment on those phases of the book. The book is excellently prepared, the illustrations are good and helpful, and the print is of such a type that it is easy to read. Various types are used; for instance, in the beginning paragraphs of each chapter all type is in capitals for two lines and, even though the sentence is not completed, the subsequent portion of the sentence is in conventional type. Free use is made of italics to stress certain phases of the text.

In the chapter devoted to roentgen diagnosis Dr. Dyke has done an excellent job. There are certain points, however, that a radiologist interested in this subject would wish to know that are not mentioned in the text. Some of these would include the information regarding the technique employed in certain examinations, whether films were made in the erect posture, lateral or horizontal, or whether they were made in extension or flexion.

One wonders about the use of terms like "lipiodolography." Is that a good name? There are so many names applied to this procedure that it would seem wise to keep them simple so that every one would be conversant with what is being talked about.

Another thing that the reviewer has in mind is the contents to be included in the book with its present title. Diseases such as leukemia, metastatic carcinoma, and Hodgkin's disease are discussed and, presumably, are regarded as surgical conditions of the spinal cord, but conditions like scoliosis and tuberculosis of the spine are not discussed. The uninitiated is somewhat at a loss to follow certain descriptive terms. For instance, a discussion is included of "intra and extradural" tumors. A further discussion is included under the status of "intra and extramedullary" tumors. These terms are not distinguished between in subsequent portions of the text and, at times in a description, for instance of a meningioma, it is stated that the lesion may occupy a position within or without the dura or arachnoid, but in other lesions the position will not be indicated. The classification as "intra and extradural" may be considered for the determination of mobility of the spinal cord. From the standpoint of roentgen diagnosis it is questionable whether one could go further than indicate that the lesion is intra- or extramedullary in its site.

The radiologist would have liked more information concerning changes in the vertebral bodies in the young individual, secondary to venous stasis produced by spinal cord tumors, vertebral anomalies in the lower spine that simulate a fracture and conditions of the spine that produce low-back pain.

The critical thoughts suggested above should not in any way detract from the importance of this excellent monograph. There are many, many features that, from the large experience and wealth of material of the authors, make this book a contribution to those interested in this subject.

Eugene P. Pendergrass

Wounds and Fractures; A Clinical Guide to Civil and Military Practice. By H. Winnett Orr, M.D., F.A.C.S., Lincoln, Nebraska, Chief Surgeon, Nebraska Orthopedic Hospital; Fellow of the American College of Surgeons, etc. Cloth. Price $5.00. Pp. 227, with 137 illustrations. Springfield, Illinois: Charles C Thomas, 1941.

There is at the present time a tremendous increase in our interest in the treatment of wounds, notably of bones and soft parts. This is due to the imminence of larger numbers of such patients injured by enemy action, and to the development of a new philosophy of the care of wounds due to the advent of chemotherapy and to the realization of the importance of the principles of immobilization and infrequent dressing as restated and crystallized by Orr. This book has a historical as well as a practical importance. The author gives in the Preface the sequential development of his ideas from his experience in the last war to the present.

The application of these principles to the treatment of osteomyelitis in civil practice is now well known and its use in war surgery has been proved by Truetta. The book is a statement of his credo as developed by discussion, case history and largely personal opinion. There are ten chapters dealing with Fundamental Principles of Fracture Treatment; The Operative Treatment of Fractures; The Treatment of Nonunion and Malunion; Compound Fractures with Infection; Fractures Involving

Joints; Nerve Injuries in Fractures; Fractures of the Pelvis, Spine and Hip; Fractures of the Foot and Hand; First Aid, Medical Care, and Physiotherapy and Conclusions.

The illustrations are roentgenograms, line drawings, photographs and charts that are helpful and adequate. The book is not a compendium of fracture treatment for the beginner, but is a statement of an important principle of the treatment of trauma to bones by one who has restated, popularized and amplified the principle of immobilization and infrequent dressing. There are, perhaps rightly, no extended references to literature but there is acknowledgment of the author's debt to Hilton, Hugh Owen Thomas, A. H. Tubby, and Sir Robert Jones.

The book is important to the clinical surgeon and to the medical historian. To the first, it is source material for a principle of treatment and to the second it may well be important as the first collected work of the author of a principle in surgical treatment that will have an important bearing on surgical practice and thought. It should be in every medical library and be read by all who treat trauma and infections of bones.

Frederick A. Coller

A Manual of the Treatment of Fractures. By John A. Caldwell, M.D., Professor of Clinical Surgery, College of Medicine, University of Cincinnati; Director of the Fracture Service, Cincinnati General Hospital, Cincinnati. Cloth. Price, $3.50. Pp. 150, with 76 illustrations. Springfield, Illinois: Charles C Thomas, 1941.

Fractures are mechanical defects produced in bone by trauma. Their management may be simple or may be impossible of perfect solution. Few reach complete knowledge from whence comes universal perfection. Any physician may be confronted with the task of their treatment. Compendiums and outlines of fracture treatment have been frequently issued to aid the needs of those whose experience and skill in treatment is meager. This manual has been designed to meet the needs of the less expert, "medical students, house officers and general practitioners." It emphasizes principles rather than methods. There are sixteen chapters dealing with the commonly seen fractures of all bones.

Following each chapter are references to books dealing with fractures in a more exhaustive manner. The illustrations, largely by Miss Mary Maciel, are well executed, informative line drawings of a schematic nature that well amplify the text. The work is an excellent abstract of the essential principles of the treatment of fractures. The author has the ability to state concisely the fundamental facts in the treatment of common and important fractures in a way that is the chief charm and value of the book. Much is told in few words. The book is small, the print and format are adequate. The book will be helpful and useful for those for whom it was written, the occasional setter of bones or the beginner whose experience and knowledge are meager but whose needs in practice demand foundations on which to work and build. To these the book is recommended.

Frederick A. Coller

The Biologic Fundamentals of Radiation Therapy: A Textbook. By Friedrich Ellinger, M.D., Research Fellow, Radiotherapy Department, Montefiore Hospital, New York City. Formerly Privatdozent in Radiology at the University of Berlin. With a Preface by Maurice Lenz, M.D., Associate Clinical Professor of Radiology, Columbia University, Director of City Cancer Hospital, Chief of Radiotherapy Departments, Presbyterian Hospital and Montefiore Hospital, New York City. English translation by Reuben Gross, M.D., Formerly Resident Physician, Radiotherapy Department, Montefiore Hospital, New York City, 1st Lieut. M.C., U.S.A. Cloth. Price, $5.00. Pp. 360, with 79 illustrations. New York: Elsevier Publishing Company, Inc., 1941.

For everyone concerned with the effects of radiation on living matter, the author has done a real service in collecting and assorting the material here presented. In that borderline field of science where biologist, physicist, chemist, pathologist, and radiation therapist meet, it is difficult for anyone to keep in touch with the literature of all phases of the work. A bibliography of 1,100 titles, all published between 1930 and 1940, attests the magnitude of the task.

The book deals not with roentgen rays and radium rays alone, but also with those sometimes overlooked forms of radiation, ultraviolet, visible, and infra-red light. After giving in considerable detail the effects of roentgen radiation on all tissues and organs, the author con-

siders the effects on inflammations and on benign and malignant neoplasms. An effort to state desirable methods of treatment for various diseases without going into sufficient detail results in a rather unsatisfactory section on actual therapy. However, the author has stated that his purpose is not to teach how to treat any particular disease, but how to understand and evaluate the biological factors underlying such treatment.

The section on ultraviolet, visible, and infrared radiation includes detailed discussions of photo-erythema and pigmentation, photosensitivity and sensitization, and vitamin D formation. In the final section are outlined the various theories which have been advanced to explain the effects of radiation, and finally, general principles are suggested for therapy.

The book is a compendium of information collected from the literature; one could wish that at times the author had been more critical regarding the work reported. In numerous instances an experimental result is stated with no intimation of the methods by which it was obtained, and no evaluation of its merits relative to other work on the same object. However, a much larger volume than the present one would have been necessary for the presentation of much critical analysis.

The bibliography, extensive as it is, has been selected mainly from generally accessible periodicals. It would have been helpful to have it arranged either alphabetically or by topics. An alphabetical author index and a good subject index are provided.

EDITH H. QUIMBY

HISTOLOGICAL STUDIES ON THE NORMAL AND THE IRRADIATED SUPRARENAL GLAND IN RABBITS: CONTRIBUTION TO THE SUBJECT OF SEASONAL CHANGES IN THE ADRENAL CORTEX AND OF THE DIFFERENTIATION OF THE CORTEX CELLS. By Olav Torgersen. Skrifter utgitt av det Norske Videnskaps-Akademi i Oslo, I. Mat.-Naturv. Klasse, 1940, No. 2. Paper. Pp. 112, with 12 tables and 7 plates. Oslo: Jacob Dybwad, 1940.

In this extensive monograph, the author points out the very slight effects of adrenal irradiation in male rabbits of varying ages.

He reviews the literature on the subject and after a short review of adrenal histology, he shows how many of the apparent histopathological changes seen and interpreted by earlier workers may be, and probably are, merely seasonal or other normal variations. Torgersen feels that incidence of low concentrations of doubly refractive fatty substances and the high incidence of the fatty or "delomorphous cells" in the Fall and Winter (when the animals are reluctant to mate) provide evidence of the seasonal variation related to the cycle of sexual activity. The component cells of the adrenal are discussed, and the author differs with the accepted views in that he does not believe the "light and dark" cells to be degenerating forms, but rather two specific cell types each with a function related to sexual activity. In his castrated animals, the "light cells" are more numerous than normal, indicating a possible attempt to compensate for the loss of the gonads.

The rabbits used were exposed to 175 kv. roentgen rays delivered through a posterior field including both adrenals. The dosage was very high, ranging from 3,000 to 6,000 r at one sitting. This dose was calculated to be as high as was compatible with life and was somewhat dependent upon age. Unirradiated animals of the same age, sex, and environment constituted the controls.

In the histopathological studies made at varying intervals after irradiation, the changes were so slight as to be recognized only with difficulty even in animals as young as one month. Occasional "vacuolar degeneration" of the cortical cells was encountered, but these same changes were found to result from irradiation of the skin, whether or not the adrenals were included in the field.

Collateral examinations of the thyroids, hypophyses, and blood cholesterol values in the irradiated animals showed no significant alterations. The testes of the irradiated animals showed slight retardation of sexual maturity.

Despite the large doses of roentgen rays to the adrenals, the seasonal variations of delomorphous cells, doubly refractive lipoids, and light and dark cells, were not interrupted or significantly altered. The author indicates clearly that these glands are radioresistant to roentgen doses far in excess of the usual clinical range.

C. L. HINKEL

BOOKS RECEIVED

THE DOCTORS MAYO. By H. B. Clapesattle. Cloth. Price, $3.75. Pp. 822, with numerous illustrations. Minneapolis: University of Minnesota Press, 1941.

Roentgen Interpretation. By George W. Holmes, M.D., Roentgenologist to the Massachusetts General Hospital and Clinical Professor of Roentgenology, Harvard Medical School, and Howard E. Ruggles, M.D., Late Roentgenologist to the University of California Hospital and Clinical Professor of Roentgenology, University of California Medical School. Sixth edition, thoroughly revised. Cloth. Price, $5.00. Pp. 364, with 246 illustrations. Philadelphia; Lea & Febiger, 1941.

Clinical Roentgenology of Pregnancy. By William Snow, M.D., Director of Radiology, Bronx Hospital; Roentgenologist-in-charge, Harlem Hospital, New York City. Cloth. Price, $4.50. Pp. 178, with 119 illustrations. Springfield, Illinois: Charles C Thomas, 1942.

Cancer of the Face and Mouth: Diagnosis, Treatment, Surgical Repair. By Vilray P. Blair, M.D., Sherwood Moore, M.D., and Louis T. Byars, M.D., St. Louis. Cloth. Price, $10.00. Pp. 599, with 260 text illustrations and 64 plates. St. Louis, Mo.: C. V. Mosby Co., 1941.

Roentgen Treatment of Infections. By James F. Kelley, M.D., F.A.C.R., Professor and Director of the Department of Radiology, Creighton University School of Medicine; Attending Radiologist, Creighton Memorial St. Joseph's Hospital, St. Catherine's Hospital and Douglas County Hospital, Omaha, and Mercy Hospital, Council Bluffs, Iowa. With the Collaboration of D. Arnold Dowell, M.D., Assistant Professor of Radiology, Creighton University School of Medicine; Assistant Attending Radiologist, Creighton Memorial St. Joseph's Hospital, St. Catherine's Hospital and Douglas County Hospital, Omaha, and Mercy Hospital, Council Bluffs, Iowa. Cloth. Price, $6.00. Pp. 432, with 122 illustrations. Chicago: The Year Book Publishers, Inc., 1942.

The Treatment of Burns. By Henry N. Harkins, M.S., M.D., Ph.D., F.A.C.S., Associate Surgeon, Henry Ford Hospital, Detroit; Formerly Instructor in Surgery, University of Chicago; John Simon Guggenheim Memorial Fellow in Surgery, 1938–1939. Cloth. Price, $6.50. Pp. 457, with 120 illustrations. Springfield, Illinois: Charles C Thomas, 1942.

Clinical Aspects of the Electrocardiogram Including the Cardiac Arrhythmias. By Harold E. B. Pardee, M.D., Assistant Professor of Clinical Medicine, Cornell University Medical College; Associate Attending Physician, New York Hospital; Consulting Cardiologist, Woman's Hospital and Methodist Hospital, etc. Fourth edition revised. Cloth. Price, $5.75. Pp. 434, with 102 illustrations. New York: Paul B. Hoeber, Inc., 1941.

Skin Grafting: From a Personal and Experimental Viewpoint. By Earl Calvin Padgett, M.D., F.A.C.S., Professor of Clinical Surgery, University of Kansas School of Medicine, Kansas City, Kansas. Cloth. Price, $4.50. Pp. 149, with 75 illustrations. Springfield, Illinois: Charles C Thomas, 1942.

Psychosurgery: Intelligence, Emotion and Social Behavior Following Prefrontal Lobotomy for Mental Disorders. By Walter Freeman, M.D., Ph.D., F.A.C.P., Professor of Neurology, George Washington University, Washington, D. C., and James W. Watts, B.S., M.D., F.A.C.S., Associate Clinical Professor of Neurosurgery, George Washington University, Washington, D. C. With special psychometric and personality profile studies by Thelma Hunt, M.D., Ph.D., Associate Professor of Psychology, George Washington University, Washington, D. C. Cloth. Price, $6.00. Pp. 337, with 81 illustrations. Springfield, Illinois: Charles C Thomas, 1942.

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INFANTILE PARALYSIS: A SYMPOSIUM DELIVERED AT VANDERBILT UNIVERSITY, APRIL, 1941. National Foundation for Infantile Paralysis, Inc. Cloth. Price, $1.25. Pp. 239, with illustrations. New York: National Foundation for Infantile Paralysis, Inc., 1941.

DAS SCHICHTBILD DER LUNGE, DES TRACHEOBRONCHIALBAUMS UND DES KEHLKOPFES. Von Kurt Greineder, Dozent für Röntgenologie an der Universität Berlin; Oberarzt der Universitätsröntgenklinik am Robert-Koch Krankenhaus. Cloth. Price, RM. 37.30. Pp. 250, with 509 illustrations. Leipzig: Georg Thieme, 1941.

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DEPARTMENT OF TECHNIQUE

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Charleston, S. C.

A ROENTGEN FILM ILLUMINATOR FOR SCIENTIFIC EXHIBITS*

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THE apparatus described briefly in this paper is a square illuminating frame with space for thirty-two 14 by 17 inch roentgenograms. This illuminator, with two 14 by 17 inch films in place, is shown in Figure 1. The illuminator can be disassembled conveniently and the parts can be placed in a packing case which is also described below.

The illuminator has several features which may be applied with economy to the construction of illuminators which are to be permanently installed in film-viewing rooms. The sources of light are eight 40 watt 48 inch and four 20 watt 24 inch daylight fluorescent lamps. The total power input (to lamps and auxiliary ballast equipment) is 480 watts. The temperature of the surface upon which the roentgenogram is placed differs by a negligibly small amount from the ambient temperature. The diffusing material upon which the films are placed is translucent Plexiglas,† a white translucent plastic which is uniform, efficient, and practically unbreakable. The material can be polished occasionally to remove fine scratches which may accumulate on the outer surfaces. The maximum and minimum luminous flux densities are 190 lumens per square foot and 130 lumens per square foot, measured by means of a Weston foot-candle meter with the measuring element held flat against the surface of the Plexiglas. Although the average luminous flux density (about 170 lumens per square foot) is lower, and the deviation of maximum from minimum luminous flux density ($190-130=60$ lumens per square foot) is higher than the standards usually specified for diagnostic film illuminators, the illumination is adequate for exhibit purposes.

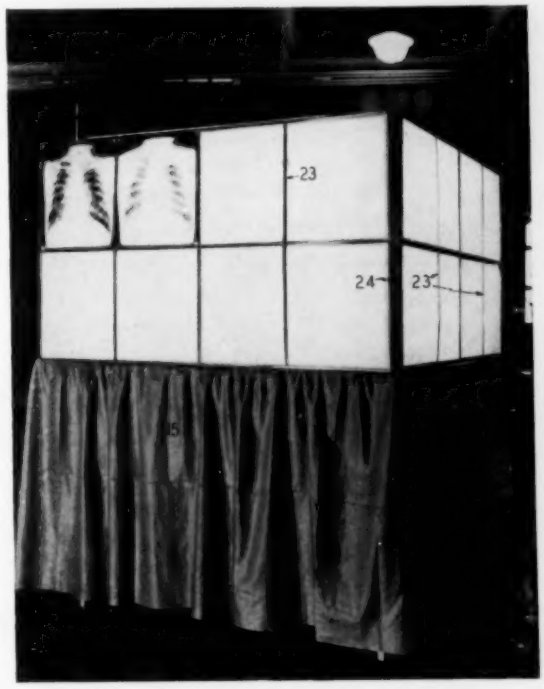

FIG. 1. Assembled roentgen film illuminator.

† "White Translucent Plexiglas No. 122–250, 0.060 inch thick," manufactured by Rohm and Haas Co., Inc., 222 Washington Square, Philadelphia.

* This device and its packing case were constructed in the Moore School Shop by M. E. Nelson and his assistant, John Pedley, for the National Tuberculosis Association. The illuminator was first used at the annual meeting of the National Tuberculosis Association held in San Antonio, Texas, May 4–8, 1941.

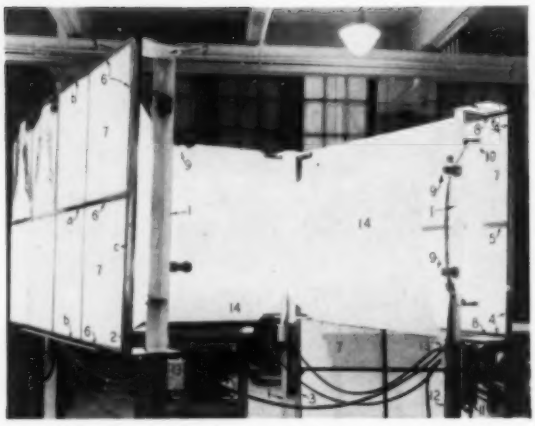

Fig. 2. One illuminating frame removed to show construction

If green fluorescent lamps were used, the average luminous flux density would be approximately 320 lumens per square foot; this value is comparable to that measured at the surfaces of the 14 by 17 inch film illuminators commonly used in a roentgen department. Furthermore, it is the opinion of some observers that green illumination of roentgenograms enables the observer to perceive smaller differences in film density than "daylight" illumination.

Brief descriptions of the structure of the illuminator and of its packing case are presented in the following paragraphs with accompanying illustrations. The total cost of materials for the illuminator and the packing case was approximately $180.00, excluding labor costs.

Figure 2 shows the illuminator with one side removed. The four legs (*1*) are pieces of extra-light weight 2 inch steel angle each 6 feet 3 inches long. Two steel plates (*2*) $6\frac{1}{4}$ inches square by $\frac{1}{8}$ inch thick are welded to each of the four legs. These eight steel plates support the four illuminating frames.

Each illuminating frame (*3*) consists of four pieces of 1 by $\frac{3}{4}$ inch heavy gauge steel angles (*4*) and two cross pieces (*5*) of $\frac{3}{4}$ by $\frac{3}{4}$ inch heavy gauge steel T. The outside dimensions of the welded rectangular frame are $33\frac{1}{2}$ by $56\frac{1}{2}$ inches. Pieces of aluminum linoleum moulding (*6*) are screwed to these steel frames. The cross-sections of the mouldings are shown in Figure 3 with letters *a*, *b*, *c*, corresponding to

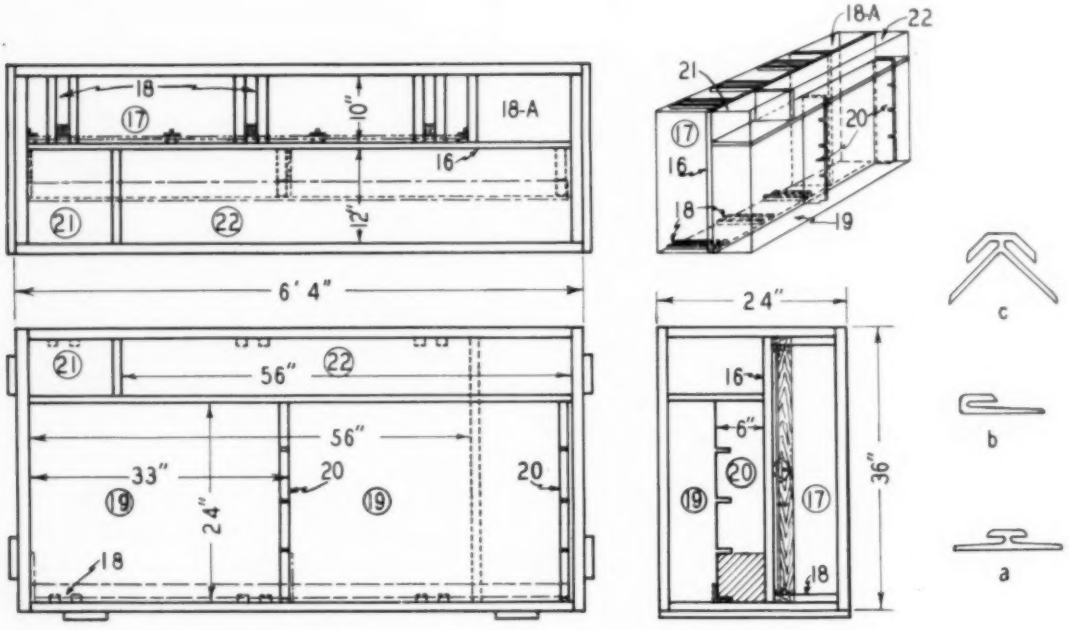

Fig. 3. Packing case for disassembled roentgen film illuminator. *Right:* cross-sections of aluminum linoleum moulding used to hold Plexiglas and roentgenograms.

similar designations of Figure 2. Before the end-moulding *c* is put in place, sheets of translucent Plexiglas 16½ by 28 by 0.060 inch (*7*) are pushed into place. The completed frames are bolted to the 6¼ by 6¼ by ⅛ inch steel plates as shown in Figure 2 (*8*). Thus the major assembling operation consists of attaching the four illuminating frames (*3*) to the four legs (*1*) by means of thirty-two bolts.

The sources of illumination are eight 40 watt 48 inch daylight fluorescent lamps (*9*) which are mounted horizontally, and four 20 watt, 24 inch daylight fluorescent lamps (*10*) mounted vertically at each of the four corners of the illuminator. The centers of the large lamps are 8 inches from the surface of the Plexiglas sheets which they illuminate.

One high power factor ballast unit for a pair of 40 watt lamps is mounted on each leg (*11*). A high power factor single lamp ballast (*12*) is mounted on each leg for a 20 watt lamp.

On each of the four legs a junction box (*13*) is mounted. This junction box contains two outlets—a standard parallel-prong, two-terminal outlet and a special three-terminal outlet.* Rubber covered conductors from each leg are connected to the next leg in counter clockwise direction. The 115 volt 60 cycle a.c. input cable is connected to four two-terminal plugs in parallel. These are inserted in the corresponding receptacles. The wiring diagram is shown in Figure 4.

The legs are painted with white enamel. Double thicknesses of Dupont white Tontine window shading (*14*) 39 inches wide and 44 inches long are stretched back of the two 40 watt lamps on each frame and attached by means of nuts and bolts to the frames at the top and bottom.

A piece of gray sateen (*15*, Fig. 1) with black elastic inserted in a loop of the material at the top is stretched around the bottom of the illuminator frame.

Roentgen films are mounted in the frame by trimming their larger dimensions to 16½ inches and slipping three edges between the Plexiglas and the aluminum moulding. The fourth (vertical) edges of each pair of films are held in place by stainless steel strips (*23*, Fig. 1) which are ground to a taper at each end so that these ends can be inserted between the aluminum moulding and the films. A stainless steel strip (*24*, Fig. 1) is slipped into the aluminum moulding at each corner of the illuminator to hide the ends of the two illuminating frames.

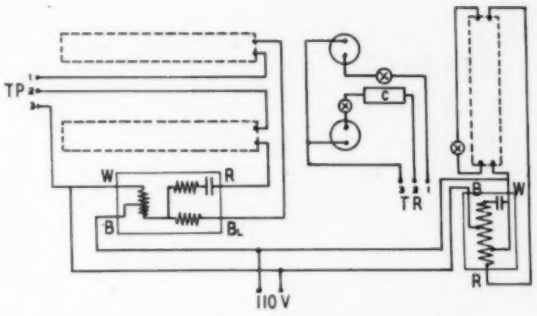

FIG. 4. Wiring diagram of apparatus attached to one leg. Dashed lines at right represent 20 watt fluorescent lamp; circles near center represent ends of two 40 watt lamps; dashed lines at left represent two 40 watt lamps. *TR* are connected to the three-conductor receptacle mounted on this leg; *TP* are connected by means of a cord to a plug which is inserted in the three-conductor receptacle in the next leg to the left. *C*, compensator; *W*, *B*, B_L, *R*, white, black, blue, and red leads from ballasts.

When the illuminator is taken apart all of its components are packed in a shipping case made of ¾ inch white pine shelving. The layout of this packing case is shown in Figure 3. The case is divided into two large compartments by the wall (*16*, Fig. 3).

The four illuminating frames are placed in one (*17*) of the compartments; each frame being separated from the others by three strips of wood placed in slots (*18*). The smaller compartment (*18-A*) on this side of the case is the receptacle of the shades, wrapped in black paper, and there is room to store roentgen films as well.

The other side of the case is divided into three compartments. The largest (*19*) is

* This fixture is "H and H receptacle No. 7092."

arranged for the four legs. Two slotted pieces (*20*) hold the legs rigidly in place. The 6 by 6 by $\frac{1}{8}$ inch steel plates of each leg are flush with one end of the box and with one of these vertical pieces; the plates are bolted in place for shipment. The smaller remaining compartment (*21*) is used for storing the input cable and a box containing nuts and bolts. All of the lamps, inserted in the corrugated paper tubes supplied with them, are surrounded by excelsior and packed in the remaining compartment (*22*). The shipping weight is 495 pounds.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

SKELETAL SYSTEM

Sommer, Franz, and Tress, Erwin. Beitrag zum Krankheitsbilde der Lipokalzinogranulomatose. (Eine besondere Form der Calcinosis universalis.) (The clinical picture of lipocalcinogranulomatosis, a special form of calcinosis universalis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, April, 1941, *63*, 205–214.

In the literature of the past few years a number of cases have been reported showing multiple tumefactions containing extensive calcium deposits in the region of the joints and they have been designated by Deutschlaender as a symmetrical progressive lipocalcinogranulomatosis and attributed by him to a systemic disease involving the synovial cavities with degeneration of the fat tissue and dystrophic calcification.

The author reports the case of a woman, aged forty-eight, who had noticed a painless, cherry-sized nodule in the left shoulder for five years. More recently she had lost much weight and felt very tired complaining of anorexia and sleeplessness. Later a nodule appeared on the left buttock and she experienced pains in the lower legs. The calcium content of the serum was 16.36 mg. per 100 cc. Roentgen studies disclosed calcified tumor masses at the left acromioclavicular joint (with a much smaller one on the right side also), in the cervical and upper dorsal areas, in the metacarpophalangeal area at the lateral aspect of the right hand, about the lesser trochanter on the right, and about the lower ends of the right humerus and of the left femur.

The masses increased rapidly in size and some recurred after operative removal. Weakness, atrophy of the musculature and anemia progressed and the patient died about five years after the first nodule was noticed. It is apparent that the calcification occurs before the tumor formation and is accompanied by a diffuse osteoporosis without deformity of the bones.

The microscopic study of a nodule showed a connective tissue capsule with cavities containing calcium and necrotic doubly refractile lipoid material surrounded by lipophagic granulation tissue with single and multinucleated macrophages. Characteristic spherocrystals were also seen.

Various theories and speculations concerning the pathogenesis are given.—*W. A. Evans, Jr.*

Pomeranz, Maurice M., and Rothberg, Abraham S. A review of 58 cases of tabetic arthropathy. *Am. J. Syph., Gonor. & Ven. Dis.*, Jan., 1941, *25*, 103–119.

The authors review 58 cases of joint disease resulting from tabes. This is usually called Charcot's disease as the first classical description of it was made by Charcot in 1868. Its most frequent site is in the knee joint; 38.5 per cent of the authors' cases were in this joint. Several joints were involved in 19 cases and in 12 the disease was bilateral. The greatest number of cases occurred in the 5th and 6th decades and in white males. Two cases were in colored females. Reported statistics in regard to the frequency of Charcot's disease in syphilis vary widely.

Neither positive neurological findings nor positive serological findings are constant in this disease. Among the authors' cases there were positive neurological findings in 48 cases, the blood test was positive in 45 per cent of the cases and the spinal fluid in 51 per cent. Since neither of these methods of examination is reliable in all cases roentgen examination becomes very important. In fact, in many cases the roentgenogram provides the first positive evidence of the syphilitic nature of the joint disease when syphilis has not been suspected.

The roentgen findings may be divided into those of early, moderately advanced and advanced stages of the disease. In the early stage the disease is hard to recognize. Perhaps the most constant finding is an accumulation of fluid in the synovial sac. This, however, is not pathognomonic as synovitis may result from any injury to the joint. A more important symptom is slight sclerosis of the bones of the joint, due to thickening of either trabeculae or cortex, more frequently the former. This

change may be slight and if in the cortex may be mistaken for a low-grade osteomyelitis.

In moderately advanced cases the effusion is greater and the synovial sac forms a tense, fluctuating tumor which obliterates the normal anatomical markings. The osteosclerosis may have progressed to the stage of intense eburnation of the bones. The cortex is irregularly thickened and there are scattered areas of periostitis. There may be small fragments of bone in close contact with the ends of the bones from which they have been detached. In cases associated with trauma a diagnosis of fracture may be made and the cause overlooked. As a result of pathologic fractures luxations are frequent and characteristic of the disease.

In the advanced stage the roentgenogram is quite characteristic. The effusion in the joint may be absorbed or replaced by a flocculent calcium deposit. This may be due to calcification of a hemorrhagic area or calcification of the synovium. The osteosclerosis is very marked and the bone intensely eburnated. The cortex is extensively and irregularly thickened. The periosteum is thickened and sometimes new bone is deposited perpendicular to the shaft, so that the picture resembles that of osteogenic sarcoma. But osteosclerosis, osteoperiostitis and cortical thickening may be lacking even in advanced cases. The joint space is narrowed and sometimes completely obliterated. Multiple fractures and luxations are very frequent and characteristic. Small fragments may appear free as joint mice or attached to the synovial membrane. In spite of the great destruction of the joint there is often evidence of repair also.

Contrary to the usual opinion, this disease is by no means always free of pain. There was pain in all but two of the authors' cases, but not so intense as would have been expected from the degree of destruction. The pain is probably due to the fact that not all of the pain fibers in the columns of Goll and Burdach are destroyed. Late in the disease after they have all been destroyed there is no pain.

In this series of cases a history of syphilis was obtained in only 39. Although some of the patients had been given long and intense antisyphilitic treatment the development of neurotrophic joints was not prevented. Whether medical treatment for syphilis is to be given depends on other factors than the joints. Surgical treatment has not proved very successful. In this group of cases 21 operations were performed on 18 patients, 2 of whom died after knee fusions. Although 11 fusion operations were performed the results were successful in only two, one in the cervical spinal column and one in the kneee. Two fusion and two reconstruction operations on the hip were all unsuccessful. It is fairly safe to say that no hip joint can be successfully fused. In one synovectomy of the knee pain became so agonizing that a low thigh amputation was necessary. Three amputations were performed because the disorganization of the joints was so great that satisfactory arthrodesis was impossible.—*Audrey G. Morgan.*

Hess, W. Röntgenologische und pathologisch-anatomische Beobachtungen bei einem Fall von Osteopoikilie. (Roentgenologic and pathological anatomic observations in a case of osteopoikilia.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Oct., 1940, *62*, 252–258.

Osteopoikilia was first described by Albers-Schönberg in 1915. About 70 cases have been reported. The areas of bone condensation are seen most frequently in the spongy portions of the bones near the joints and most rarely in the skull and vertebrae. No definite clinical symptoms have been observed. The condition is a hereditary recessive characteristic. The case here reported is remarkable in that it was complicated by acute myeloid leukemia and that a complete postmortem study could be made. The patient was a twenty-seven year old woman with high fever and hemorrhages from the skin and mucous membranes. The white cell count varied from 8,000 to 40,000 per cu. mm. Death occurred after a six weeks' illness. The lesions of osteopoikilosis were numerous throughout the skeleton except in the skull, sternum, and spine. Microscopic studies showing the character of the lesions are described and illustrated. The findings of Schmorl and Brücke were confirmed. There was thought to be no causal connection between the osteopoikilosis and the leukemia and no influence of the one on the other.—*W. A. Evans, Jr.*

Bullo, Enzo. Un raro caso di tumore osseo. (An unusual case of bone tumor.) *Radiol. med.*, Feb., 1941, *28*, 110–111.

A case is described in a young man of twenty-one in whom a tumor developed on the anterolateral surface of the upper end of the tibia. There was no history of trauma. Roentgen examination seemed to show osteosarcoma and he was given several roentgen treatments with-

out any result. He was then sent to the author, whose roentgen examination also showed a picture similar to that of osteosarcoma but with some atypical features.

Amputation of the limb showed a grayish tumor with some hemorrhagic cysts in it. It was found to have originated in the musculature and to have broken through the cortex of the bone later. Histological examination showed that it was a malignant rhabdomyoma.

Such tumors, originating in the soft parts and extending to bone secondarily, are quite rare. The patients are generally young. Differentiation in these cases is very difficult for the tumors are generally confused with bone sarcoma or sarcoma of the fascia.

The treatment of choice in malignant rhabdomyoma is surgical.—*Audrey G. Morgan.*

MASSERINI, ATTILIO. Aspetti radiografici dei tumori giganto-cellulari delle ossa trattati con roentgenterapia. (Roentgen appearance of giant cell bone tumors treated with roentgen irradiation.) *Radiol. med.*, Feb., 1941, *28*, 84–91.

There has been a good deal of discussion in recent years of the differentiation of benign giant cell tumors of bone from sarcoma, and the possibility, now generally admitted, of their undergoing malignant degeneration. There has been little or no discussion, however, of their appearance after roentgen treatment and of the effectiveness of this treatment in this form of tumor.

The author therefore describes and illustrates with roentgenograms 5 cases which he has had under observation for some years. The details of treatment, which varied somewhat in the different cases, are given. It is hard to tell clinically when these cases improve or are cured, for the swelling of the bone does not recede. But there is a marked change in the roentgen picture after treatment. Before treatment the bone presents the appearance almost of a multilocular cyst, though in some cases the bone inside the thin shell of cortex seems to be completely destroyed. After treatment there is a very evident recalcification of the cortex, and of the trabeculae if the bone design has been preserved, until finally the bone appears solid and resistant again or there may even be an intense eburnization. If the tumor had broken the cortex and extended into the soft parts this part of the tumor becomes calcified also.

These changes, however, take place very slowly. In some cases, as in the author's first one, the condition even seems to grow worse at first; this is known as Herendeen's paradoxical phenomenon and consists of an apparent continued decalcification of the cortex and extension into the soft parts, which is followed later by the recalcification described above. The process of recalcification after it begins may continue for three or four years. The apparent exacerbation at first therefore should not discourage the physician, but on the contrary the series of roentgen treatments should be repeated several times.—*Audrey G. Morgan.*

WISE, R. A. Lateral dislocation of the head of the radius with fracture of the ulna. *J. Bone & Joint Surg.* April, 1941, *23*, 379–381.

Monteggia emphasized the frequent occurrence of dislocation of the head of the radius with a fracture of the shaft of the ulna. The radial dislocation is usually anterior but may be posterior or lateral; the ulnar fracture is usually in the upper third of the shaft but may be anywhere in the shaft.

The author reports a case in which the ulnar fracture was in the upper third and the radial dislocation was lateral in type. The important diagnostic point from the roentgen standpoint is the relation between the head of the radius and the capitellum. If this relationship is preserved no dislocation exists.—*R. M. Harvey.*

SIEGLING, JOHN A. Growth of the epiphyses. *J. Bone & Joint Surg.*, Jan., 1941, *23*, 23–36.

It is generally accepted that growth of the diaphysis takes place at the cartilage columns in the epiphseal plate, and that certain growth cartilages contribute more than others to longitudinal growth. Proof of these facts was in part due to the proper interpretation of transverse lines observed in long bones following the clinical and experimental administration of phosphorus. The pathology of these transverse opacities was speculative until 1927, when Eliot demonstrated that they were the result of a bony lattice work, produced by the enlargement and cross branching of the trabeculae, which at times illustrated even transverse bridging. Lines have also been observed in cases of congenital syphilis, in severe illness (particularly scarlet fever), in lead poisoning, and after ingestion of certain drugs. In cases where phosphorus has been administrated intermittently, multiple lines have been observed.

Siegling's attention has been attracted for

some time to the appearance of lines in the epiphyses in roentgenograms of children as a result of disease or phosphorized cod-liver oil medication. Since interpretation of the radiopaque lines in the diaphyses has thrown light on shaft growth, he thought that study of similar lines occurring in epiphyses might clarify the question regarding their growth.

Growth activity in the epiphysis is confined to the articular cartilage by microscopic evidence. In the roentgenograms of children, radiopaque lines due to lead, bismuth, phosphorized cod-liver oil, etc., Siegling observed, assumed very definite patterns. These patterns were dependent upon the method of growth of that particular epiphyseal cartilage. In practically all cases new bone seemed to have been added to the epiphysis everywhere except at the epiphyseal cartilage. He found in roentgenograms of knees of growing children in whom surgical fusion of the knee had been performed that, with removal of the articular cartilage and fusion of the osseous elements of the adjacent epiphyses of the femur and tibia, the epiphyseal cartilage plates did not grow farther apart. The vertical diameter of the combined epiphyseal nucleus did increase. This is additional proof that growth of the epiphysis takes place by proliferation of the articular cartilage followed by endochondral ossification, and that epiphyseal cartilage is a negligible factor in longitudinal growth of the epiphysis, if it contributes at all.—*R. S. Bromer.*

Wilmoth, C. L. Osteochondromatosis. *J. Bone & Joint Surg.*, April, 1941, *23*, 367–374.

The author describes this entity as an uncommon one in which multiple intra-articular cartilaginous bodies are found as free bodies or attached. The etiology has not been definitely established. During a ten year period the author saw 6 cases of osteochondromatosis involving the knee joint, 2 of the elbow joint and 1 of the hip joint. The patients were all males varying in age from twenty-six to sixty-four.

He summarizes the 9 cases and includes illustrative films showing involvement of the hip, elbow, and knee joints respectively. Most of the cases complained of some degree of pain, swelling and limitation of motion of the involved joint. All cases but one were subjected to operative removal of the loose bodies with partial synovectomy. No recurrences were noted but the follow-ups were limited to a four year period, and in view of the slow-growing tendencies of these masses the author does not feel that this is a sufficient length of time to warrant any claims of cure.

The size and number of the joint bodies was responsible for the objective findings and the subjective symptoms are those usually attributed to arthritis. The diagnosis is confirmed by roentgenograms.—*R. M. Harvey.*

Stauffer, H. M., Arbuckle, R. K., and Aegerter, E. E. Polyostotic fibrous dysplasia with cutaneous pigmentation and congenital arteriovenous aneurysms. *J. Bone & Joint Surg.*, April, 1941, *23*, 323–334.

Recently fibrous dysplasia involving multiple bones has been differentiated from osteitis fibrosa cystica. The authors present a case with unilateral fibrous displacement of bone, cutaneous pigmentation and multiple arteriovenous aneurysms in the extremities showing the most marked bone changes.

The patient was a male, aged nineteen, admitted because of protrusion of the left eye and a recent history of brownish pigmentation of this eyelid, lacrimation and shooting pains in the eye. An injury to the left arm in childhood was followed by enlargement and bowing. Protrusion of the left eye first occurred at the age of five following trauma. At this time a roentgen diagnosis of "bone cysts" was made. The left femur was fractured three times. Enlargement of the left upper arm occurred following minor traumata. The veins of the left upper arm were prominent since early childhood. A pathological diagnosis of osteitis fibrosa cystica was made on a biopsy of the left forearm at the age of eleven.

The important physical findings were bony enlargement of the left frontal region and left jaw, increased diameter of the left arm, prominence and coxa vara deformity of the left hip, increased diameter of the left thigh, and genu valgum of the left knee with left cavus and equinus deformity of the foot. There was a large pigmented area over the lower posterior thorax, to the left of the midline. Thrills and bruits were present over the left upper arm and leg.

Roentgenograms showed bilateral lesions of the bones, more advanced on the left. The skull showed marked thickening of the frontal bone with replacement of the frontal sinus. The shaft of the left humerus was expanded, the cortex thinned, and the medulla showed irregular trabeculations intermixed with lucent areas suggesting cysts. Similar changes were found

in the bones of the hand. The pelvis, left side, was asymmetrical and distorted and showed similar rarefaction. The left femur was likewise involved. The blood phosphatase level was markedly elevated and the calcium and phosphorus normal. Biopsy of the bones affected showed a diffuse fibrous dysplasia which had destroyed normal bone architecture and growth.

In the differential diagnosis osteitis fibrosa cystica generalisata, osteitis deformans, Ollier's dyschondroplasia and neoplasm were ruled out. The authors felt that their case fitted the picture of polyostotic fibrous dysplasia as described by Lichtenstein. Their case had the unusual complication of multiple congenital arteriovenous aneurysms. They feel that both changes are due to a congenital disorder of mesoderm.

Cases of polyostotic fibrous dysplasia with skin pigmentation have been described previously.—*R. M. Harvey.*

SACHS, MAURICE D. Familial brachyphalangy. *Radiology*, Nov., 1940, *35*, 622–626.

Brachyphalangy is a condition in which one or more of the phalanges are short and thick. It has rarely been reported as a familial condition. The author discusses a family in which 32 members, 15 females and 17 males, had brachyphalangy involving only the middle phalanges of the fifth fingers. Roentgenograms are given showing these phalanges short and broad and curved outward. The little fingers as seen in photographs curved outwards.

The anomaly was transmitted by both males and females. There were no cases of mongolism or idiocy in the family. It was at one time thought that this condition occurred only in mongols or idiots. Roentgenograms of the feet and sella turcica were normal. The above cases cannot have been caused by amniotic bands as it is highly improbable that such bands would affect only the middle phalanges of the fifth finger in five generations.—*Audrey G. Morgan.*

SCOTT, W. Epiphyseal dislocations in scurvy. *J. Bone & Joint Surg.*, April, 1941, *23*, 314–322.

Scurvy is a state of hypovitaminosis C with a tendency to hemorrhages and secondary anemia. Skeletal changes appear early in the disease although often unrecognized in the mild cases. Osteoclastic bone resorption is accelerated throughout the skeleton, especially at the metaphyseal ends of the long bones. Recent workers have shown that the epiphyseal separations occur through the zone of temporary calcification, which is represented on the roentgenograms by the white line at the end of the metaphysis. This zone is vulnerable to trauma by reason of its high calcium content. Hemorrhages occur beneath the periosteum simultaneously with the development of lattice fractures. The periosteum is stripped from the shaft. This stripping away of the periosteal cuff predisposes to epiphyseal separations.

The infant skeleton possesses remarkable adaptability in adjusting itself to the deformities occurring with epiphyseal separations. New bone is laid down by periosteal bone formation mainly on the side of the shaft toward which the epiphysis is displaced and the epiphysis becomes centered again at the end of a broadened metaphysis. Through secondary bone resorption the shaft is reduced to its original size and shape.

In the case of more marked dislocations a new shaft is formed within the periosteal covering which remains when the shaft escapes through a large rent. The extruded shaft is absorbed.

The order of frequency of the sites of these fractures is lower femur, upper humerus, costochondral junction and lower tibia.

According to the author only one roentgenographic sign is conclusive, the white line at the metaphyseo-epiphyseal junction with a zone of rarefaction on the shaft side of this white line. Calcification in subperiosteal hematomas only occurs with healing and requires ten days to become visible on the films. Traumatic dislocations can be differentiated by the absence of the white line.—*R. M. Harvey.*

HUDSON, R. T., HUCHERSON, D. B., and ORTNER, A. B. Complete bilateral epiphyseal separation of the upper humeral epiphysis due to scurvy. *J. Bone & Joint Surg.*, April, 1941, *23*, 375–378.

This is a case report of an unusually severe case of scurvy in which complete bilateral separation of the upper humeral epiphyses occurred. The authors were not able to find a similar case reported in the literature subsequent to 1917.

Their patient was a female aged nine months, who had been denied vitamin-containing foods since birth. The child had exhibited generalized tenderness and swelling of the extremities since

the age of six months. Roentgenograms of the entire body showed wide separation of both upper humeral epiphyses and calcified subperiosteal hemorrhages about the humeri, femora, tibiae and fibulae. Marked increased density of the zones of temporary calcification was present.

The epiphyseal separations of the humeri were treated by manipulation under a general anesthetic and fixation in abduction in a double body spica. Check-up films following adequate vitamin therapy after a two month interval showed normal relationship of the upper humeral epiphyses to the shaft. Dense callus was present about the shafts of both humeri at their upper ends.—*R. M. Harvey.*

Bell, Joseph C. The roentgen-ray examination in individuals suffering from low back and sciatic pain, with examples of some lesions in which errors in diagnosis may readily be made. *Radiology*, Oct., 1940, *35*, 449–458.

Cases of pain in the lower part of the back and sciatic nerve are described and illustrated with roentgenograms. Roentgen examination with lipiodol was made in these cases and showed various types of lesions, including fractured vertebrae and herniated intervertebral discs.

Close cooperation between neurologist and roentgenologist is necessary in such cases. Contrast medium should not be injected into the spinal canal unless neurologic examination indicates that this method is advisable.

The author prefers the use of 2 cc. lipiodol for the examination and has never had any bad results from its use. A number of films should be made covering all the regions in which pathological lesions are suspected. He seldom makes less than eight exposures in the anteroposterior direction and usually at least two in one of the lateral positions.

In some cases it has been impossible to distinguish between defects caused by hypertrophied ligaments and those caused by herniated intervertebral discs. In a large percentage of cases herniated intervertebral disc is accompanied by a hypertrophied ligamentum flavum. But there may be a hypertrophied ligament at one level and a herniated disc at another. Hypertrophy of the ligamentum flavum is a definite entity but the major symptoms are probably caused by some associated lesion in most cases.

In traumatic cases that do not do well and develop low back pain roentgen study of the spinal canal is indicated.—*Audrey G. Morgan.*

Nichols, Bernard H., and Nosik, William A. Myelography with the use of thorium dioxide solution (thorotrast) as a contrast medium. *Radiology*, Oct., 1940, *35*, 459–466.

When thorium dioxide was first injected into the subarachnoid space there were severe reactions and even some deaths. But with the development of thorotrast—an alkaline buffered suspension of thorium dioxide in a protective colloid dextrin—these difficulties were overcome. Thorotrast is very well borne.

Even this substance, however, caused considerable irritation of the meninges and it seemed desirable to remove it after the myelograms were made. Animal experiment showed that this could be done without injury by forced drainage of the cerebrospinal fluid. When the method was used in human beings quantitative analysis of the removed spinal fluid and of 1 cc. of the thorotrast injected showed that more of the substance was eliminated than of any other radiopaque substance that had been used before. The quantitative recovery was from 70 to 90 per cent. The radium equivalent of the alpha and gamma radiation from the residual thorotrast was far below the minimal toxic level.

It is evident that this method of examination should not be used in subarachnoid block but only in non-obstructive or partially obstructive lesions in the canal. But these are the lesions in which diagnosis is most difficult and surgery most hopeful.—*Audrey G. Morgan.*

Garland, L. H. The effect of iodized oil on the meninges of the spinal cord and brain. *Radiology*, Oct., 1940 *35*, 467–476.

Animal experiment has shown that lipiodol (fresh iodized poppy seed oil) can be injected into the subarachnoid space of the spinal canal without danger. The oil remains in the patient's cerebrospinal system indefinitely and may be mistaken on later roentgen examination for serious lesions. The author in collaboration with Morrissey has examined such patients years after the injections and found no clinical or neurological symptoms caused by the presence of the oil. He believes the use of lipiodol justified in cases in which it is indicated. Indiscriminate use of any contrast medium is not justified.

In the discussion of this series of papers on spinal examination Dr. Camp said that contrast examination of the spinal canal might show herniated discs but the symptoms were apt to be caused by some associated pathological condition. Tumors in the cervical or thoracic canal may cause low back pain. He emphasized the necessity for thorough neurological examination before introducing any opaque medium into the spinal canal. In using iodized oil he prefers 5 cc. to the 2 cc. recommended by Dr. Bell. He has not been able to rule out small non-obstructing tumors definitely without the use of 5 cc. of oil. Practically all protruded discs are associated with hypertrophied ligamentum flavum, so the roentgen differentiation is not important. His only objection to thorotrast is that it is an intense irritant to soft tissues, and in doing lumbar puncture it is often impossible not to get some of the medium in the epidural space. He emphasized the necessity for using fresh lipiodol which is transparent and of a very faint yellow color.

Many of the reactions from lipiodol are due to the use of a deteriorated oil. If the oil has any brown tinge it should not be used. He thinks lipiodol more accurate than air for such work.—*Audrey G. Morgan.*

ADELSTEIN, LEO J. Spinal extradural cyst associated with kyphosis dorsalis juvenilis. *J. Bone & Joint Surg.*, Jan., 1941, *23*, 93–101.

Adelstein reports a case of extradural spinal cyst associated with kyphosis dorsalis juvenilis and reviews 16 other cases collected from the literature. He concludes that the clinical picture of an adolescent with progressive paraplegia, together with erosion of the vertebral bodies in the mid-thoracic region as seen in kyphosis dorsalis juvenilis, is pathognomonic of an extradural spinal cyst. All cases of kyphosis dorsalis juvenilis should be carefully examined for evidence of spinal cord compression and for roentgenographic evidence of enlargement of the spinal canal. The deformity seen in this condition in association with extradural cyst results from venous stasis secondary to the presence of the cyst. The etiology of these associated disease processes is unknown. Early surgical intervention with removal of the intraspinal cyst usually results in complete cure with relief of all signs of compression of the spinal cord and prompt arrest of the kyphotic deformity.—*R. S. Bromer.*

CLEVELAND, MATHER. Tuberculosis of the spine. *Am. Rev. Tuberc.*, Feb., 1940, *41*, 215–231.

This report deals with 330 joints of tuberculous involvement studied in the Sea View and St. Luke's Hospitals in New York; 52.4 per cent of these were spinal lesions; 17.2 per cent concerned the knees; 10.6 per cent the hips; 7.0 per cent the sacral iliac joints; 4.8 per cent the tarsal joints; 1.8 per cent the ankles and 0.6 per cent the symphysis pubis—thus, 94.4 per cent were weight bearing joints. The remaining 5.5 per cent, non-weight bearing joints, included the shoulders, 0.9 per cent; the elbows, 3.0 per cent and the carpal joints, 1.6 per cent; 58.6 per cent were males; 41.4 per cent were females. With consideration of age, the highest incidence was found in the third decade though close approximations were found in the first two decades.

Rarely were the joint lesions diagnosed within six months of the onset of symptoms; the average duration of symptoms prior to complete medical analysis was three years and three months. The symptoms included root pains, tenderness and slight curvatures. Muscle spasms were usually noted, while roentgenographically (in those cases having symptoms for six months or longer) the findings varied from merely narrowing of the joint space to actual destruction of adjacent vertebrae and abscess formations. Not infrequently, surgical procedures had been undertaken with consideration of gallbladder disease or because of hernias or retroperitoneal tumors, in most instances of which the correct diagnoses would have been revealed by roentgenography of the spine.

Of the various levels, the highest incidence of involvement was between the seventh thoracic through the fifth lumbar. Rarely were the cervical or sacral segments involved. Forty-eight of the 203 patients showed some evidence of cord pressure. The greatest number of these had lesions at the seventh or eight thoracic vertebrae. Abscess formations occurred in 86 per cent of the St. Luke's group (30 patients) and 68 per cent of the Sea View group (173 patients); 13 per cent of the St. Luke's group showed sinuses while 22 per cent of the Sea View group showed them (with the mortality of 50 per cent). Spontaneous fusion resulted in 23.6 per cent of the spinal lesions but the average time requirement was almost seven years—

practically twice as long as required after surgical intervention. In most cases, a modification of the Hibbs operation was performed. Of these patients, 55.7 per cent were returned to their homes within twelve to eighteen months with excellent results; 25.1 per cent died.—*A. A. de Lorimier.*

Mayer, Ernst G. Über Wirbelveränderungen bei Rückenmarkstumoren. (Vertebral changes associated with spinal cord tumors.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, May, 1941, *63*, 293–299.

Changes in the vertebrae are not sufficiently recognized as a sign of spinal cord tumor. The first sign is a widening of the intervertebral foramen observed particularly in hour-glass neurinomas. In one instance this is well shown in an oblique view of the cervical spine with the lesion between the fourth and fifth vertebrae. The foramina are demonstrated with difficulty in the dorsal area, but more easily in the lumbar spine. Another sign is the widening of the spinal canal as recognized in the sagittal view by the method of Elsberg and Dyke. The lesion should be located at the highest level of expansion as the lower limits of the tumor are indeterminate by this method. Changes occur which are seccondary to spinal fluid block and there may be expansion of the canal one or two vertebrae above the actual site of the tumor. A third sign hitherto undescribed has been observed repeatedly in tumors of the cervical area. It consists of an atypical position of the vertebrae producing an angular kyphosis at the site of the lesion. Three cases illustrating this sign are presented. One was in a fifty-three year old woman with an angulation between the second and third cervical vertebrae, and myelography showed a complete block at this level. The second concerned a fourteen year old child with an angulation between the fourth and fifth segments. The myelography showed a complete block just above this level and at operation a glioma was demonstrated. The third case was a thirty-six year old woman with a kyphosis at the fourth and fifth cervical level; myelography and later operation disclosed the lesion, a neurinoma, at this level. Such a change in the intervertebral disc may be falsely interpreted as an inflammatory process, but in such a case there would be a more evident involvement of the vertebral body. Sometimes a lesion of the soft tissues may permit a subluxation of the bodies, i.e. a slight spondylolisthesis, but the appearance is different from the angular kyphosis associated with an intraspinous lesion.—*W. A. Evans, Jr.*

Benárd, Miklós, and Kovács, Ákos. Osteoarthropathie hypertrophiante mit Veränderung des endokrinen Systems. Zur Genese des Syndroms Marie-Bamberger. (Hypertrophic osteoarthropathy with changes in the endocrine system. Concerning the genesis of the Marie-Bamberger syndrome.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Nov., 1940, *62*, 316–321.

This condition is described as a symmetrical thickening of the distal epiphysis of the long and short tubular bones as the result of periosteal new bone formation usually in association with chronic pulmonary or cardiac disease. The case report concerns a twenty-five year old man who at the age of sixteen began to notice enlargement of the distal phalanges of the hands and feet. At the age of nineteen he suffered an acute polyarthritis and the following year was hospitalized with endocarditis and ascites. Loud systolic and diastolic murmurs were audible and the heart was enlarged to the left. The hands and feet were enlarged with drumstick deformities of the fingers and toes and there was a remarkable hyperextensibility of the distal phalangeal joints. Very thick laminated periosteal shadows were demonstrated roentgenologically in the long bones and there was osteophyte formation about the joints. The sella turcica was small (42 sq. mm. vs. a normal of 75–115 sq. mm.) with bony bridging of the clinoid processes.

The osteoarthropathy was thought not to be acromegalic in character and to have no connection with the cardiac condition. The blood sugar curve fell to 56 mg. per 100 cc. one and a half hours after the ingestion of 40 gm. of glucose with mild symptoms of a hypoglycemia. This finding together with the small sella and the evidence of irregular bone development led to a diagnosis of hypophyseal dysfunction.—*W. A. Evans, Jr.*

Wichtl, Otto. Über Erniedrigung der letzten Lendenbandscheibe und das Vorkommen lumbosakraler Übergangsbandscheiben. (Narrowing of the last lumbar intervertebral disc and the occurrence of transitional lumbosacral discs.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Oct., 1940, *62*, 229–247.

The fifth lumbar vertebra and its disc are subject to anatomical variations. Sacralization or a tendency to the sacral type of vertebra is called the ascending or cranial type. On the other hand, the 25th vertebra may not be included in the sacrum leading to an increase in the number of presacral vertebrae (lumbarization, i.e. caudal or descending type). Transitional forms are common (10 per cent) and of these 9 per cent are lumbarization. Their clinical significance lies in the question of their presence as a basis for pain. The last lumbar disc is wedge-shaped and in youth measures 6 mm. thicker at the anterior aspect. The angle formed by the adjacent bony surfaces is 10–27° with an average of 17° in youth and 6–29° with an average of 16° in adults. The discs undergo physiological age changes as early in life as the second decade and there is a progressive narrowing with respect to the vertebral bodies throughout life. This tends to be more marked anteriorly and results in a greater mobility which leads to sclerosis of the adjacent bony surfaces and lipping.

No close correlation exists between the roentgen appearance and the severity of the patient's complaint and very advanced changes may be present without any complaint. Rarely must tuberculosis and osteomyelitis be considered in the differential diagnosis and other inflammatory conditions are occasionally responsible for a narrowed disc.

In transitional anatomical forms (sacralization) there may be narrowing of the disc on a degenerative basis (Schmorl) with arthrosis and bony sclerosis, but the narrowing may also be purely anatomical in character. Beginning about the fifteenth year, the sacral vertebrae begin to fuse with disappearance of the intervertebral discs, and this occurs in a cranial direction and is generally complete at twenty-five to thirty years. When there is sacralization of the fifth lumbar vertebra, a corresponding narrowing of the lumbosacral disc may be expected. The absence of sclerosis, lipping, and other degenerative changes in the adjacent bone and about the articular facets speaks for the anatomical character of the narrowing. However, it is thought that these transitional discs are more subject than normal ones to degenerative changes.—*W. A. Evans, Jr.*

STEHR, L. Das Wirbel-Hämangiom. (Vertebral hemagioma.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Sept., 1940, *62*, 179–188.

Primary tumors are much less common than secondary metastases in the spine and of the primary tumors the greater number are benign. Hemangiomas are by far the most common primary tumors. In Schmorl's Institute there was an incidence of 10.7 per cent in 3,829 spines examined. Many of these hemangiomas were very small and would not have been demonstrable during life. The author has collected 14 cases, some of them symptomless and 3 were verified by autopsy. The roentgen appearance is characteristic and permits an unequivocal diagnosis of the lesion. Although the lesions are benign in the sense that they do not metastasize, some of them are progressive not only producing pressure on but destroying the adjacent bone. While some of the trabeculae are destroyed, others become thickened to take up the support of the body and, as might be expected, these are chiefly the vertical trabeculae. In addition to the altered trabecular markings there may be an expansion of the body, usually laterally but occasionally posteriorly so as to narrow the neural canal. The lower dorsal and upper lumbar bodies (i.e. those with the richest blood supply) are most commonly involved and the cervical region only very rarely. The lesion is predominantly monostotic and such was the fact in the author's 14 cases, but careful pathologic study may show microscopic lesions in other vertebrae. There was a predominance of females in the ratio of 9:6. Cases have not been described below the age of twenty. That the involved bodies are quite solid is shown by the observation of a compression fracture in an adjacent healthy vertebra. The instance of a compression fracture involving a hemangiomatous vertebra was observed and the subsequent observation over a five year period showed normal healing and no change in the character of the lesion. In the differential diagnosis actinomycosis and Paget's disease may rarely offer difficulty.—*W. A. Evans, Jr.*